INHERITANCE OF RESISTANCE TO *RHIZOCTONIA SOLANI* KUHN IN SNAP BEANS (*PHASEOLUS VULGARIS* L.)

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ABSTRACT

The inheritance of resistance to Rhizoctonia solani Kühn in snap beans was studied using crosses between 4 resistant lines, Cornell 2114-12, PI 226895, PI 165426, and B 4096, and 3 susceptible ones, 'Harvester', 'Hawaiian Wonder', and 'Manoa Wonder'. Disease evaluations were made in a greenhouse in artificially infested beds with known inoculum. Disease was rated on a scale of 1 (resistant) to 5 (susceptible) based on the size and depth of lesions on 2 week old seedlings.

Disease ratings for the resistant parents ranged from 1.08 to 1.69 and for the susceptible parents from 3.93 to 4.88. The average disease ratings for the F_1's were intermediate between the parents with low variance, while in the F_2 progeny segregated into all 5 classes. There was, however, no segregation in progenies from crosses among the resistant lines, all the F_1 and F_2 progeny being resistant.

The 3 most resistant lines, Cornell 2114-12, PI 226895, and PI 165426, all apparently carry the same genes for resistance and differ by 3 pairs from the 2 most susceptible lines, 'Harvester', and 'Hawaiian Wonder'. 'Manoa Wonder' already has 1 pair of genes for resistance and thus differs by only 2 pairs from the 3 most resistant lines. B 4096 appears to be still segregating for resistance.

The genes act quantitatively, so that individual plants with at least 4 genes for resistance are considered resistant (class 1 or 2) while those with 3 or less genes are considered susceptible

(classes 3, 4, and 5).

The broad sense heritability was estimated as between 73.4 and 91.4% and the narrow sense heritability was estimated to be higher than 68.3%. Reciprocal crosses responded similarly, thus giving no evidence of cytoplasmic factors being involved.

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INTRODUCTION

Diseases caused by soil borne fungi are recognized in all bean growing areas of the world as a persistent and little understood production problem, usually referred to as root rot. *Rhizoctonia solani* is a major component of this complex, along with *Fusarium solani* f. sp. *phaseoli*, *Pythium ultimum*, and *Thielaviopsis basicola*. *R. solani* causes many types of diseases in beans including seed decay, pre- and post-emergence damping-off, hypocotyl lesions and cankers, as well as root rot. The effects are often noticed as an uneven stand due to the death of seedlings which are attacked prior to or immediately after emergence.

Control of this pathogen by cultural and chemical methods has been difficult and unpredictable, often failing when conditions for disease development are optimum. The development of cultivars with genetic resistance has the potential to be a promising method of control.

The mechanism of inheritance of resistance to *R. solani* in beans has not yet been determined. This has delayed the development and release of resistant cultivars. The objective of this study is to establish the mode of inheritance of resistance to *R. solani* in snap beans. Since *R. solani* is a pathogen of beans in many parts of the world including Hawaii and Sri Lanka, this study is of both fundamental and practical importance.

LITERATURE REVIEW

The Pathogen - Rhizoctonia solani

R. solani was first reported by Julius Kühn in 1858 from diseased potato tubers. Since then, this soil fungus has gained a reputation of being a widespread, enormously destructuve, versatile pathogen. Although it has been the subject of hundreds of research papers, there is some confusion and disagreement over the taxonomy and nomenclature of this fungus as well as many aspects of its pathology (Menzies, 1965). It does not form spores during its vegetative growth and was originally believed to possess no sexual stage. It was, therefore, classified as order Mycelia sterilia of the class Fungi Imperfecti. However, in 1903, Rolfs recorded the development of the sexual (perfect) stage on the surfaces of diseased stems during prolonged periods of warm wet weather. The sexual spores, however, corresponded to the taxonomic characteristics of the class Basidiomycete. In 1956, Donk proposed the name *Thanaptephorus cucumeris* for the sexual stage. Various authors classified its perfect stage in several different genera and names such as *Hypochnus cucumeris* (Frank, 1903), *Corticium vagnum* (Burt, 1903), *Hypochnus solani* (Burt, 1916), *Pellicularia filamentosa* (Rogers, 1943), *Ceratobasidium filamentosa* (Olive, 1957) were proposed but were not acceptable for both taxonomic and nomenclatural reasons. It is the asexual (imperfect) stage of the fungus that causes disease in plants, and at present, *Rhizoctonia solani* Kühn is accepted as the valid name for the asexual stage and *Thanaptephorus cucumeris* (Frank) Donk is favored for its sexual stage (Menzies, 1965).

R. solani can be easily identified by the following vegetative characteristics (Parmeter, 1970).

1. A rapidly growing mycelium of relatively large diameter (>5μ), branching near the distal septum of hyphal cells, often at right angles in the older hyphae,
2. A constriction of the branch hyphae at the point of origin,
3. A septum of the branch hyphae near the point of origin,
4. Multinucleate cells in actively growing hyphae,
5. A prominant septal pore apparatus.

Garret (1938) distinguished soil fungi as soil inhabitants and soil invaders. He considered R. solani to be a soil inhabitant, capable of surviving in the soil as a saprophyte. It is also a primitive or unspecialized parasite with a wide host range. This wide host range confers the advantages of abundance and wide distribution upon the parasite. R. solani probably causes more different types of diseases on a wider variety of plants, over a larger part of the world, and under more diverse environmental conditions, than any other plant pathogenic species (Baker, 1965). It commonly attacks the juvenile tissues (Garret, 1938), and is best known for the black scurf disease in potato and as a prime cause of damping off of seedlings and other symptoms in many vegetables such as snap beans, peas, lettuce, cabbage, tomato, and pepper (Baker, 1965). It also causes important root diseases in crops such as wheat and oats (Hassan, 1956), rice (Hashioka, 1951), sugar beets (Downie et al., 1952), and cotton (Lutra and Vasudeva, 1941).

In snap beans R. solani causes seed decay, seedling blights (pre and post emergence damping off), cotyledon lesions, hypocotyl lesions and girdling of seedling stems, and root rot. In the field and in seed beds disease is often noticed as an uneven stand due to the death of seedlings which were attacked prior to or immediately after emergence (Zaumeyer and Thomas, 1957). Sometimes, pods that touch the ground are also infected (Schroeder, 1953). Diseases caused by soil borne fungi are recognized in all bean growing areas of the world as a persistent and little understood production problem (Steadman, 1974). R. solani is undoubtedly responsible for a major share of this kind of disease (Menzies, 1965). Zaumeyer and Thomas (1957) estimated that losses of snap beans from R. solani diseases vary from season to season and from one area to another but losses of 5 to 10% are common and may be much higher when conditions for disease development are favorable. Moore and Conover (1955) estimated that infections exceeded 90% in some areas of Florida.

Host-Parasite Physiology

1. Strain Specialization

Although the morphological characters of R. solani are identical in many hosts, it has been reported by Storey (1941), Blair (1942), Leclerg (1941), Richter and Schneider (1953), and Flentje and Saksena (1957) that heterogeneous strains differing in their nitrogen metabolism, tolerance to acid and alkali, as well as in pathogenicity to differing hosts, exist. Flentje (1957) found that some strains were limited to hosts of a single family while others were capable of

attacking a wide range of hosts. Christou (1962) found in comparative studies that a root infecting strain isolated from other hosts when tested on bean attacked only its hypocotyl, producing numerous small, brown individual lesions.

2. Mode of Host Invasion

The process of seedling infection by R. solani was reported by N. T. Flentje and his associates at the Waite Agriculture Research Institute of the University of Adelaide. Kerr and Flentje (1957) observed three distinct stages in the infection of a radish hypocotyl:

a. The hyphae become firmly attached to the cuticle of the seedling hypocotyl,
b. Complex, dome shaped multicellular structures known as infection cushions are formed,
c. The host is then invaded by means of fine infection pegs (attenuated hyphal tips) produced from the underside of the infection cushion.

Christou (1962) reported that the infection process of a bean hypocotyl is similar to that described for radish. Strains of R. solani not pathogenic to a particular species of seedling failed to become attached to the hypocotyl and to organize infection cushions thereon. Dodman and Flentje (1960) felt that fungal hormones may be involved in the development of infection structures.

An essential condition for the infection by R. solani is the production of attractive exudates by the germinating seeds, the

hypocotyl, and the roots (Kerr, 1956). These exudates contain sugars and amino acids in nutritionally significant concentrations as well as vitamins and a wide variety of other organic substances (Rovira, 1965). In three varieties of Phaseolus vulgaris, Schroth and Cook (1964) found a significant correlation between the amount of exudation from germinating seeds and susceptibility of young seedlings to preemergence killing by R. solani. Exudates of younger seedlings caused greater stimulation of growth of R. solani than exudates from older seedlings, due to a decline in the material exuded (De Silva and Wood, 1964). By growing the fungus on cellophane over exudates from host and non-host species, Kerr and Flentje (1957) observed that infection cushions were produced on the films overlying host but not non-host exudates, but hyphal penetration of the cellophane did not follow. However, penetration did follow when strips of host hypocotyl epidermis were placed on host exudate in agar; on this the normal 'infection' process occurred just as on the hypocotyl of a living seedling. However, on host hypocotyl epidermal strips placed on agar without exudate, attachment of the fungus and growth took place, but infection cushion formation and penetration failed to follow. From this evidence, they concluded that both a host type of epidermal surface and a host type of exudate are essential for the normal sequence of invasion and infection. On the basis of similar experiments, De Silva and Wood (1964) postulated that specificity to attack by R. solani is determined by the nature of the epidermis and the exudate of the host seedlings.

Flentje (1957) listed four ways in which infection failures may occur in host parasite combinations involving R. solani:

a. Hyphae fail to attach to the host surface,

b. Hyphae become attached to and grow over the host surface, but fail to form infection cushions,

c. Infection cushions are formed, but penetration by infection pegs is prevented by thickening of host cell walls as an active response to attempted infection,

d. Penetration succeeds but hyphae are killed by a hypersensitive reaction resulting in small necrotic spots.

It is not yet established whether penetration by infection pegs from infection cusions is primarily by mechanical pressure or by the enzymatic destruction of host constituents. Adequate evidence to support either view is lacking (Bateman, 1965). Several investigators favored the mechanical pressure hypothesis of ingress (Christou, 1962; Gonzales and Owen, 1963; Van Etten et al., 1966). However, R. solani has been shown to produce a range of enzymes such as cutinases (Linskens and Haage, 1963), pectinases (Christou, 1962; Bateman, 1963), cellulases (Kohlmeyer, 1956; Garret, 1962), and proteases (Van Etten and Bateman, 1963). These enzymes are capable of destroying various components of the host. Dodman and Flentje (1965) felt that cutinases could degrade the cuticle beneath the infection structures. The proteases may function in cell wall degradation and in the process of maceration of host tissue in addition to breaking down the structural components of the host cell protoplast. The pectinases and the cellulases aid the pathogen to spread through the host tissue by both intercellular and intracellular penetration. The alteration and

destruction of cellulose within the host has been demonstrated in Rhizoctonia infected tissues (Bateman, 1965). The pectin degrading enzymes have been identified as polygalacturonase and pectin methyl esterase.

Several workers observed discoloration and injury of host tissue in advance of penetration by certain R. solani isolates (Kerr, 1956; Wyllie, 1962). To account for these observations these workers postulated that the non-enzymatic phytotoxic products are produced by the fungus. Sherwood and Linberg (1962) studied the phytotoxin production of R. solani using two isolates, No. 245 and 282, grown on two media, glucose-nitrate medium and corn meal sand medium. They found that a phytotoxin was produced only by the isolate No. 282 and then only in the corn meal sand medium, but not in the glucose-nitrate medium. They reported that this phytotoxic molecule had phenolic and glycosidic properties. This finding suggests that production of phytotoxin is not a constitutive property of the fungal metabolism but rather depends on the substrate on which the fungus is growing. This phytotoxic substance was later identified as or closely related to O-nitrophenyl-B-D-glucoside (Sherwood, 1965).

R. solani can also enter its hosts directly through stomata and wounds. No infection cushions are formed, the hyphae simply enter through the stomata or wound, decreasing in diameter if necessary. Cracks in the seed coat of bean seeds provide a ready means of entrance, resulting in destruction of the plumule and the radicle (Webster and Goth, 1965).

Following penetration of the cuticle and cell wall of the bean

hypocotyl the infection pegs enlarge inside the epidermal cells and then invade the cortex both intracellularly and more abundantly, intercellularly, advancing longitudinally and transversely through the middle lamella and the intercellular spaces, enveloping the cortical cells (Christou, 1962). One of the first symptoms of R. solani infection is the browning of host cells as a result of intracellular penetration and subsequent killing of the protoplasm (Boosalis, 1950; Christou, 1962). In advanced infections the development of the fungus in the tissues is so profuse that the collapsing cells become compressed. This explains why the symptoms on the hypocotyl below the soil level frequently appear as dark brown, deeply sunken lesions.

Van Etten et al. (1966) observed that the expansion of R. solani lesions on a bean hypocotyl is a very rapid process. They characterized lesion development by three stages which they designated young, intermediate, and mature. The young stage is characterized by a water soaked appearance, but the lesion remains colorless or light fawn in color. Lesions in the intermediate stage of maturation differ from those of the young stage only in that the lesion surface has become brown to dark brown in color and in some instances there is slight macroscopic evidence of tissue collapse within the lesion area. Mature lesions are characterized by a dry appearance of the lesion surface and a collapse of the invaded susceptible tissues. Lesions assume the characteristic concave appearance and the surface coloration ranges from dark brown to brick red. In all 3 stages, endopolygalacturonase activity was much higher than cellulase activity. They felt, however, that both pectolytic and cellulolytic activities would be associated

with disease since the pathogen invades both intercellularly and intracellularly. Following ingress, hyphal invasion of the cortex proceeds from one cell layer to another. Where severe lesions develop, the fungus often enters the vascular tissues and the pith. As there was no indication of any wound periderm formation by the host (Christou, 1962; Van Etten et al., 1966), it seems evident that factors other than structural barriers are necessary to account for the induced resistance which results in rapid lesion limitation. These workers supported the contention that the presence of cell wall materials resistant to enzymatic attack rather than the inactivation of the cell wall degrading enzymes may be an important factor in induced resistance by the host.

3. Host Response to Infection

The violent disturbance of the normal host metabolism following infection serves as a warning to the outlying tissues, which then respond with the production of phytoalexins and perhaps also other chemical agents (Garret, 1970). Phytoalexins are non specific, phenolic compounds with fungistatic properties. They are synthesized de novo or greatly increased in the plant as a defense mechanism to pathogenic attack. Plants belonging to many families produce phytoalexins and many investigators feel that this is the plant's most effective response to challenges by unspecialized parasites. Smith et al. (1975) isolated four phytoalexins - phaseollin, phaseollidin, phaseollinisoflavan and kievitone from bean hypocotyls with R. solani lesions. These four phytoalexins were concentrated in the lesion

areas per se and accumulated with time. However, they found only kievitone and phaseollin attained concentrations high enough to create an antifungal environment during the early and presumably critical stages of pathogenesis. They felt that if any involvement of phytoalexins occurred in disease resistance via lesion delimitation, then kievitone and phaseollin appeared to be primarily responsible among the four phytoalexins. Morris and Smith (1978) also reported similar findings. Ayer et al. (1976) proposed a model for host-pathogen interactions in which they postulated that in incompatible interactions which produce a resistant reaction, the growth and the spread of the pathogen is inhibited by a rapid build up of toxic concentrations of phytoalexins surrounding the lesions, whereas in a compatible interaction which produces a susceptible reaction, the pathogen colonizes new tissues before the phytoalexin concentration reaches high enough levels to inhibit the fungus.

Bateman and Lumsden (1963) found that up to a certain critical age all the tissues of a young seedling are juvenile and susceptible to infection by R. solani. In beans, the hypocotyls are highly susceptible during the first two weeks, moderately resistant during the third week, and resistant thereafter. They indicated that the changes in resistance were associated with elongation and maturation of the hypocotyl and concomitant changes in the pectic substances and calcium content. The calcium content of bean hypocotyls increased from 0.38% at 4 days to 1.92% at 16 days. Calcium was also found to accumulate in and around the R. solani lesions which suggests that resistance may develop from the conversion of pectins in the middle lamella to calcium pectate

which is not hydrolyzable by the pectic enzyme polygalacturonase secreted by the pathogen. This enzyme is believed to play an important role in the intercellular spread of the pathogen within the host. Bateman and Daly (1966) reported that bean hypocotyls bearing R. solani lesions exhibited increased respiratory rates at all stages of lesion development. Their studies indicated a striking change in respiratory metabolism which preceeds or accompanies limitation of lesion size. Smith et al. (1975) felt that the localized accumulation of kievitone and phaseollin to antagonistic levels, in conjunction with calcium pectate formation as well as perhaps several as yet unrecognized facets, may help explain the limitation of R. solani to restricted lesions on bean hypocotyls in the absence of an observed physical barrier. Bateman (1965) speculated that although the formation of calcium pectate is probably only one of a complex of factors involved in resistance to R. solani, the possibility exists that, if young seedlings are furnished with adequate calcium, the period of high susceptiblility might be reduced.

Environmental Factors Affecting R. solani Infection

R. solani can survive, grow, and remain pathogenic in soil under a wide range of environmental conditions (Sinclair, 1965). Formerly, pathogenicity was thought to depend mainly on the intrinsic properties of the pathogen and host with environmental factors rarely of more than secondary importance (Shephard and Wood, 1963). However, there is evidence now that the environment plays more than a secondary role. The effect may not always be directly on the fungus, but may be indirectly on the host plant, as with soil temperature and soil reaction (Baker

and Martinson, 1965).

1. Soil Temperature

The early work of Richards (1923) and Leach (1947) established that disease severity was not solely a function of fungal growth. Even though R. solani grows more rapidly in culture at about 30°C, the disease it causes on potatos, peas, and beans is most severe at about 18°C (Richards, 1923). Richards suggested that, although the higher temperature is best for the growth of the fungus in agar, the enzyme or group of enzymes responsible for tissue destruction is secreted more abundantly or reacts more with the host tissue at 18°C. There was, however, no evidence to support such a hypothesis. The fungus grows fairly well at 15 to 20°C, but most seedlings mature more slowly at this temperature, making the period of susceptibility longer than at higher temperatures when seedlings grow and mature faster and 'escape' or 'grow away' from disease. Peltier (1916), however, reported that higher termperatures near 30°C favored disease development by R. solani in carnations.

Significant observations relating temperature and disease incidence were made by Leach in 1947. The disease incidence did not correspond closely with the growth rate of either the host or the pathogen at different temperatures. Instead, the disease incidence was inversely related to the ratio between the coefficient of velocity of seedling emergence (CVE) and the growth rate (G) of the fungus. When the disease incidence was compared with this ratio at various soil temperatures, the disease incidence increased as the ratio decreased. The strain of Rhizoctonia used by Leach grew on solid media from 8 to 40°C. Rapid growth

was observed between 25 and 30°C. With cool temperature crops such as spinach and sugar beets, there was a low incidence of preemergence damping-off at low temperatures (4 to 12°C), where host growth was favored over pathogen growth and the ratio CVE/G was high. At higher soil temperatures especially 20 to 30°C, the relative growth rates favored the pathogen, the ratio CVE/G was lower, and preemergence damping-off was severe. A warm season crop, watermelon, showed opposite results: extensive seed decay and preemergence damping-off were observed at soil temperatures below 25°C with a low CVE/G ratio, but disease incidence was low at 30 and 35°C with a much higher CVE/G ratio. Leach found, in all of the combinations he tested, that the CVE/G ratio accurately predicted the incidence of damping-off. Beach (1949) confirmed Leach's results and conclusions in experiments with tomatoes, cucumbers, spinach, and peas.

Various strains of R. solani have different temperature optimums for disease development. Three strains isolated from cotton had three different optimum temperatures (Hunter et al., 1960). Bean isolates from Wisconsin caused the greatest disease development from 16 to 24°C while North Carolina isolates incited disease best at 28°C (Baker, 1961). Lettuce damping-off strains were most active near 8°C (Shephard and Wood, 1963), whereas strains causing bottom rot of lettuce were most active above 24°C (Townsend, 1934).

2. Soil Moisture and Aeration

Water and air in the soil are important factors for disease potential (Baker and Martinson, 1965). Damping-off of seedlings occurred at

soil moisture concentrations between 20 to 80% of saturation (Blair, 1943). The disease was less severe at or near saturation due to lack of sufficient aeration. There was an almost linear inverse relationship between 30 to 80% saturation (Bateman, 1959). The pathogen is most active near the soil surface with the entire activity confined to the upper 2 inches of soil (Papavizas, 1973). High relative humidity near the soil surface as a result of overcrowding and overwatering is very conducive to disease development in seed beds (Garret, 1944). The fungus can spread rapidly from hypocotyl to hypocotyl above the soil surface and the unobstructed growth of mycelia enables the pathogen to spread more rapidly than it can through the soil, so that seedlings are killed by hypocotyl infection in advance of root infection.

3. Soil Reaction

In general, most strains of R. solani grow quite well over a broad pH range and certainly within the pH limits of most agricultural soils and host tissues (Baker and Martinson, 1965). Many isolates prefer a pH range of 5.8 to 8.1, a neutral reaction being optimum for highest growth (Blair, 1943). There is no evidence at present to say that pH affects the inoculum potential or disease potential of R. solani. There is a possibility that the pH of the rhizosphere soil solution may affect enzyme production or enzymatic activity of the fungus (Bateman, 1963). Certain mineral ions such as calcium and magnesium have been linked to disease development, and since availability of these ions is related to the soil pH, the pH could indirectly affect the disease potential through mineral nutrition.

Methods of Study

R. solani does not form asexual spores such as conidia. Sexual spore (Basidiospore) production is rare, difficult to obtain in culture, and lacking many isolates (Sinclair, 1965). The fungus produces sclerotia which are hard compact masses of fungal tissue that enable it to survive long periods in the soil. Some isolates, however, produce few, if any, sclerotia in culture. Infection by basidiospores and sclerotia appears to be rare and unpredictable. For this reason, Sinclair (1965) suggested that mycelia obtained from hyphal tip cultures should be used in pathogenicity tests.

Standard procedures for isolation of plant pathogenic fungi can generally be used to isolate R. solani from infected plant tissues (Sinclair, 1965). Placing infected tissue pieces in isolation in water agar minimizes competition, allows R. solani to grow away from most competitors, and facilitates hyphal tip isolation (Parmeter, 1965). The fungus grows on a wide range of solid and liquid media but grows most rapidly on potato-dextrose-agar and in Richards medium (Sinclair, 1965). Some of the media which have been used to grow cultures of R. solani are cornmeal-sand (Gibson et al., 1961; Prasad and Weigle, 1970; Deaken and Dukes, 1975), vermiculite mixed with nutrients (Varney, 1961), potato dextrose broth (Bateman, 1961), potato dextrose agar (Christou, 1962), sterile oats (McLean et al., 1968), synthetic Czapek's medium (Prasad and Weigle, 1970), and wheat grains (Dickson and Boettger, 1977). These media can be infested with the mycelia to produce a pure culture. When fully colonized, the cultures can be used to inoculate plant parts, or infest soil or any other medium.

Sinclair (1970) listed several advantages of using pure cultures for pathogenicity tests:

a. Effective control of inoculum levels,
b. Precise measurement of fungus effects,
c. Production of severest disease condition possible,
d. Reproduction of results,
e. Elimination of stimulatory and antagonistic effects of other organisms.

The disadvantages of using pure cultures are:

a. Conditions are not comparable to field conditions,
b. Disease is likely to be more severe than under natural infestations.

Pathogenicity tests with R. solani have usually been undertaken in greenhouse. Sterilized compost, soil, sand, peat, vermiculite, perlite, or combinations of these have been used to grow the test seedlings to be inoculted (Christou, 1962; Dickson and Boettger, 1977).

Disease evaluations under field conditions have been undertaken only to a limited extent. This is because of the variability of pathogen distribution in the field and interactions with other pathogens in the soil, both of which may cause unpredictable and erratic disease development (Sinclair, 1965). Although using field soil with or without artificial infestation approximates the disease situation in the field, it has several disadvantages:

a. Reproducible results are difficult to obtain because of variability from one soil sample to another,

b. Data may be appropriate only for the area or areas from which the soil was sampled,

c. The test may not represent the severest disease situation possible.

A method for quantitative determination of R. solani in soil was described by Ko (1972). This method uses a selective medium which allows only R. solani to grow and suppresses the growth of other microorganisms. The medium consists of 1g K_2HPO_4, 0.5g $M_gSO_4.7H_2O$, 0.5g KCl, 10mg $FeSO_4.7H_2O$, 0.2g $NaNO_2$, 0.4g gallic acid, 90mg Dexon, 50mg Chloramphenicol, 50mg Streptomycin, and 20mg agar in 1 liter of distilled water. The antibiotics chloramphenicol and streptomycin inhibit growth of bacteria and actinomycetes, while the fungicide Dexon, which is not inhibitory to R. solani, prevents the growth of pythiaceous fungi. Gallic acid and sodium nitrite are added as carbon and nitrogen sources because they are stimulatory to the growth of R. solani but are inhibitory to or unmetabolizable by many other fungi. By plating a designated amount of soil from an infested field in this medium, Ko was able to determine the population of R. solani in terms of the number of propagules of R. solani per 10 grams of soil. Ko found 1 to 9 propagules per 10 grams of soil in infested potato and bean fields. He found that even this small amount of inoculum has a high disease inducing potential, causing almost 90% preemergence damping-off of beet seedlings in soil inoculated with 9 propagules per 10 grams of soil in the laboratory.

Loss of pathogenicity after prolonged in vitro culture has been reported. Isolates from rotted tomato fruit lost their infectivity after 2 months in culture (Shrouder and Proventi, 1961). The use of newly isolated fresh cultures of the fungus was recommended for inoculation. Castanho and Butler (1978) reported that R. solani usually is very stable and isolates kept in culture for as long as 30 years on PDA retain a high level of virulence. Nevertheless, they have occasionally observed cultures in a state of decline. These sick cultures were white to tan in color, irregular in appearance, produced few or no sclerotia, and had a slower growth rate than healthy cultures. Because of the degenerative nature of this disease, they named in the Rhizoctonia decline. The agent responsible for Rhizoctonia decline has not been resolved. They were able to cure this disease and recover healthy cultures by merely taking hyphal tip isolations from diseased cultures. Based on these results, they stressed the value of hyphal tip isolation before and during research with R. solani especially with regard to maintenance of virulence.

Control of R. solani

1. Cultural Practices

Control of R. solani by cultural practices is difficult because the fungus can survive in the soil for long periods of adverse environmental conditions in the form of thick walled mycelia and sclerotia (Garret, 1956). Transmission by infected seeds is also possible (Baker, 1947; Leach and Garber, 1965). At times, early seeding, shallow planting of seed, and proper management of crop residues have been effective in

reducing damage (Leach and Garber, 1965). With some exceptions, crop rotation is ineffective as a control measure because of the broad host range of the pathogen (Zaumeyer and Thomas, 1957). R. solani infection in cotton was usually more severe following plantings of alfalfa, pasture legumes, and cowpeas. Rye is also an undesirable crop to use in rotation because it builds up the R. solani population. However, Wellman (1932) and others have indicated that the isolate of R. solani from crucifers is relatively specific to crucifers, so cabbage can be rotated with grain crops or legumes to control Rhizoctonia head rot. Corn and sorghum have also been reported effective as rotation crops to control R. solani (Papavizas, 1973).

In bean fields in Maryland, Papavizas (1973) observed the highest inoculum density of R. solani in July immediately after ploughing under the first planting. Rhizoctonia problems were more severe during the second bean planting in July than during the first planting in May. The inoculum density was lowest in March, 1 to 2 months before the first bean planting.

Henis et al. (1979) studied the factors affecting suppression of R. solani in soils. They noticed that the pathogenicity and growth of R. solani was suppressed in some soils initially infested with this pathogen and planted repeatedly with radishes. Soils differed in their capacity to develop suppression under monoculture but there was a greater increase in soil lytic properties and populations of the fungus Trichoderma spp. in suppressive soils than in conducive soils. Members of Trichoderma spp. are well known for their parasitic attack on R. solani. Pieczarka and Abawi (1978) studied the interactions of

Fusarium solani f. sp. phaseoli, Pythium ultimum, and R. solani on bean root rot. They studied the pathogens singly and incombination in uniformly infested, pasteurized, bean field soil in a controlled environment. They found a synergistic relationship between P. ultimum and F. solani. Both in combination produced a very high incidence of hypocotyl and root disease. No such interaction was observed between R. solani and F. solani. However, R. solani significantly reduced the severity of disease incited by P. ultimum when the two were combined, suggesting an antagonistic relationship between them. Very high incidence of disease was noticed when all three pathogens were present.

2. Chemical Methods

R. solani can be eradicated from soil to be used in a greenhouse by steam sterilization or by broad spectrum chemicals such as methyl bromide and vapam (Baker, 1957). In the field, it can be controlled by the fungicides pentachloronitrobenzene (PCNB) and captan in a 1:1 mixture by weight at the rate of 8 to 10 lbs/A (Leach et al., 1959). Ideally, the fungicide is best applied to the seed furrow (Bell and Owen, 1963). However, PCNB only strongly suppresses the growth of the fungus rather than kills it (Ko, 1972). Also PCNB accentuates diseases caused by Pythium aphanidermatum in soybeans while decreasing damping-off due to R. solani (Gibson et al., 1962). The PCNB selectively acted on R. solani, which permitted more damage by the unaffected Pythium. The effectiveness of chemical treatment to prevent R. solani infection in snap bean is unpredictable, often failing when conditions are optimum for disease development (Deaken and Dukes, 1975).

Tolmoff (1962) pointed out another possible disadvantage of fungicidal control of R. solani. He found that bean varieties differ considerably in tolerance to PCNB, so that some bean cultivars, especially certain processing types, exhibit poor root development and stunting of seedlings at the concentration of PCNB required to prevent infection.

These limitations associated with chemical control combined with the high cost have stimulated interest in the development of genetically resistant cultivars in many crops including snap beans.

3. Genetic Resistance

Although crop plants differ considerably in susceptibility to infection, it has been difficult for plant breeders to develop cultivars resistant to R. solani (Leach and Garber, 1965). This is attributed to the nature of the parasitism, the wide host range, and the lack of sharp differentiation among strains of the pathogen.

Luthra and Vasudeva (1941) were unable to find any resistant varieties of cotton. Richter and Schneider (1953) encountered the same situation with wild species and varieties of Solanum. Some potato varieties, however, had some resistance and this was correlated with germination vigor, rapid growth, vigorous sprouts, extensive root and stolon production, regenerative power, and an undefined resistance of shoots to invasion of R. solani. Improvement of the resistance of sugar beets to R. solani was reported by Downie et al. (1952). They found Nebraska 525, a selection, definitely more tolerant to infection than its parents. A selection of large seeded lima bean with good tolerance to root rot was reported by Kendrick and Allard (1952). Other crops in

which varietal resistance has been reported are gladiolus (Creager, 1945), lettuce (Poole, 1952), rice (Hashioka, 1951), and cabbage (Williams and Walker, 1966). In cabbage, the resistance appeared to be inherited as a monogenic dominant character. Barksdale (1974) could not find high levels of resistance to Rhizoctonia fruit rot among commercial varieties of tomato. From over 3,000 tomato PI lines tested, R. L. Clark (1978) reported useful resistance in 3 lines. He considered all lines with more than 1/4 of the fruit surface rotted as susceptible. His study indicated that resistance is determined by a single, dominant gene. These findings, however, did not agree with that of Barksdale (1974) who had reported that resistance is inherited as a polygenic character.

A search for genetic resistance to R. solani disease in snap beans started in the 1950's. Yerks and Freytag (1956) believed that resistance in Phaseolus coccineus was superior to that in P. vulgaris. From more than 600 bean lines screened in artifically infested greenhouse soil between 1965 and 1967, McLean et al. (1968) identified 12 lines with resistance, PI 181954, PI 156414, Venezuela 54, PI 165426, PI 165435, B 3866, PI 318696, PI 318697, PI 318699, PI 318700, PI 179934, and PI 165426 x Alabama #1 F_2. These lines exhibited a very high emergence and low incidence of hypocotyl and root lesions. Five more lines were reported by Prasad and Weigle in 1970. They were plant introduction accessions 226896, 109859, 163583, 174908, and 300665. They reported that highly resistant lines were free of infection or had only water soaked lesions, while susceptible lines exhibited seed decay, damping-off, and dark brown large lesions in the hypocotyl.

They indicated that resistance was associated with colored seed coat, fast germination and seedling emergence, and woodiness of the hypocotyl. They felt that inadequate growth nutrients and fungistatic compounds in the resistant bean hosts retarded infection during the early phases. They listed the following characteristics as factors affecting the host-parasitic interaction:

a. Cracking and germination of the bean seed,
b. Exudates of the host,
c. Rate of maturation of the host tissue,
d. Phytoalexin production by the host,
e. Toxin production by the fungus,
f. Calcium content of the host tissue.

Wallace and Wilkinson (1973) noticed that R. solani resistant bean lines had small seed size. A small, colored seeded bean, Cornell 2114-12, derived from a cross between P. vulgaris and P. coccineus, was resistant to 4 soil borne pathogens Fusarium, Pythium, Thielaviopsis, and Rhizoctonia (Bravo et al., 1969).

Deaken and Dukes (1975) reported that resistance was highly heritable, and controlled by few major genes. They felt that resistance was associated with colored seed and were unable to obtain resistant white seeded lines. They released a resistant breeding line B 4096 with purple seed.

The seed coat pigments of beans are phenolic glycosides (Deaken and Dukes, 1975). A high phenolic content of bean seed coats has been related to host resistance to pathogenic infection (Statler, 1970).

Some important phenolic compounds in plants that are involved in disease resistance are lignins, flavanoids, and phytoalexins. Biosynthesis of the phenolic compounds in the plant takes place through the shikimic acid pathway. Deaken and Dukes (1975) suggested that the genetic system that controls the pigment formation in the seed coat also controls the production of other important phenolic compounds such as phytoalexins so that the white seed coats are always associated with disease susceptibility. If this is the case, it is unlikely that breeding methods will be effective in altering the association between colored seed and resistance to R. solani.

Further work on the relation between seed coat pigments and resistance was reported by Prasad and Weigle (1976) and Wyatt (1977). The former demonstrated the presence of higher levels of amino acids and the absence of phenolic compounds in the extracts of white seed coats. They observed that the nutritional requirements in terms of amino acids for pathogenesis are not satisfied by resistant black seeded lines. Extracts of white seed coats stimulated growth of R. solani but the extracts of black seed coats contained phenolic compounds that inhibited growth of R. solani. However, they pointed out that not all colored seeded beans are resistant to R. solani. They suggested that the susceptibility to R. solani may be due to a larger percentage of seed coats of white and red seeded cultivars that cracked during initial hydration of the seed and therefore protected the germinating seed from infection for only a very short period of time. The seed coats of the black seeded cultivars, however, adhered tightly to the cotyledons and many times remained over them until after emergence, protecting them

from R. solani. The degree of damping-off in resistant cultivars could be increased by artifically cracking the seed coat before germination. Approximately 2/3 of the resistance was attributed to the seed coats remaining intact until they are burst open by the hypocotyl. Prasad and Weigle, therefore, suggested that the differences in levels of resistance to R. solani were primarily due to the integrity of the seed coat, and secondarily to the phenol content of the seed coat. They suggested that the greater importance of seed coat integrity compared to color may permit the development of white seeded cultivars with a moderately high level of resistance.

Wyatt (1977) reported that colored seed coats had greater dry weight and thickness than white seed coats and that the white seed coats were more permeable to water. The white seeds, therefore, absorbed water more rapidly than colored seeds. The slower absorption of water by colored seeds permitted more uniform swelling of cotyledons, thereby reducing seed coat and cotyledon cracking.

In spite of these reports. Dickson and Boettger (1977) were able to obtain white seeded Rhizoctonia resistant F_2 segregants from a cross between Cornell 2114-12 (colored seed, resistant) and G_4 (white seed, susceptible). In contrast to the findings of Deaken and Dukes (1978), they reported that resistance to Rhizoctonia is independent of seed color and is quantitatively inherited, although they were unable to specify the number of genes that were involved.

Hagedorn and Rand (1978) have also released white seeded bean breeding lines with resistance to R. solani. These lines RRR77 and RRR83 also have resistance to several other soil pathogens involved in the Wisconsin bean root rot complex.

MATERIALS AND METHODS

Testing for Resistance

All tests for resistance were done in greenhouse benches with a controlled amount of a known inoculum. The inoculum was prepared by a modification of the cornmeal-sand method of Deaken and Dukes (1973), as follows:

Formula for cornmeal-sand medium

1. 2043 g. (4.5 lbs.) of air dry silica sand. Sand should be free of organic matter and preferably of medium grain size.
2. 103 g. commercial yellow cornmeal, 5% by weight of the sand.
3. 215 ml. distilled water, 10% by weight of the sand and cornmeal.

This amount is prepared in one 2800 ml. Fernback flask.

Preparation of the cornmeal-sand inoculum

1. Mix dry sand and cornmeal thoroughly.
2. Add distilled water slowly while mixing.
3. Place moist cornmeal-sand medium in Fernback flask and plug with cotton. (The materials can be mixed adequately in the flask, if desired).
4. Sterilize in an autoclave for one hour at 15 psi (121°C internal temperature).
5. Allow flask to cool, but before cold agitate vigorously to loosen the medium.
6. Allow flask to stand for at least 24 hours at room temperature

(24°C).

7. Sterilize again for one hour at 151 psi (121°C internal temperature).
8. Infest the medium with 10 small blocks of potato dextrose agar containing R. solani and agitate to disperse throughout the medium.
9. Incubate the infested flask cultures for 8 days at a temperature of 24-26°C. Agitate flask every other day to prevent the formation of large lumps and caking.

Maintenance of R. solani Inoculum Source

R. solani isolated from infected bean hypocotyls from Poamoho Agriculture Experiment Station is maintained on susceptible bean plants growing in the greenhouse. This is to prevent a possible decline in pathogenicity during invitro culture. When the cornmeal-sand medium is to be infested, the fungus is isolated from the infected plants by planting pieces of infected hypocotyl tissue on water agar for 24-28 hours and then transferring the hyphal tips of the fungus to a plate containg potato dextrose agar. The identification of R. solani was based on the hyphal characteristics reported by Parmeter and Whitney (1965) and also by the nature of the symptoms formed on infected bean hypocotyls. Three to four days later, the agar containing the fungus is ready to be used for infesting the medium.

Testing Conditions

All testing was done in specially constructed wooden benches filled 25 cm. deep with a 75:25 mixture of No. 2 vermiculite and perlite.

A new mixture was used for each test. Seeds to be tested were placed 2 cm. apart in a 5 cm. deep furrow in the moistened germinating medium. The seeds were then covered with the infested cornmeal-sand inoculum at the rate of 400 g. inoculum per 100 cm. furrow length, as suggested by Deaken and Dukes (1975). This level of inoculum gave very good seperation between resistant and susceptible lines (See Table 3, p.38).

Soil temperature in the benches ranged between 21 and 31°C which is within the range established for pathogenicity of R. solani (Leach, 1947; Beach, 1949). The benches were watered lightly as needed every other day.

Fourteen days after sowing, the plants were dug up and scored individually for their disease reaction on a scale of 1 to 5 as follows (Figure 1):

Class 1: Completely free of lesions or with only water soaked lesions or superficial blemishes in the epidermis of the hypocotyl

Class 2: Having a few small to medium, brown surface lesions, some cortical damage, but no vascular damage

Class 3: Having a few large, deep sunken brown lesions, involving the vascular tissue

Class 4: Having many large, deep sunken dark brown lesions involving the vascular tissue and almost completely girdling the hypocotyl

Class 5: Seedlings dead, having either failed to emerge or with the

cotyledons and terminal buds infected; or alive but severely stunted with a completely girdled hypocotyl

Classes 1 and 2 can be classified as resistant and classes 3, 4, and 5 as susceptible.

Pathogen Variability

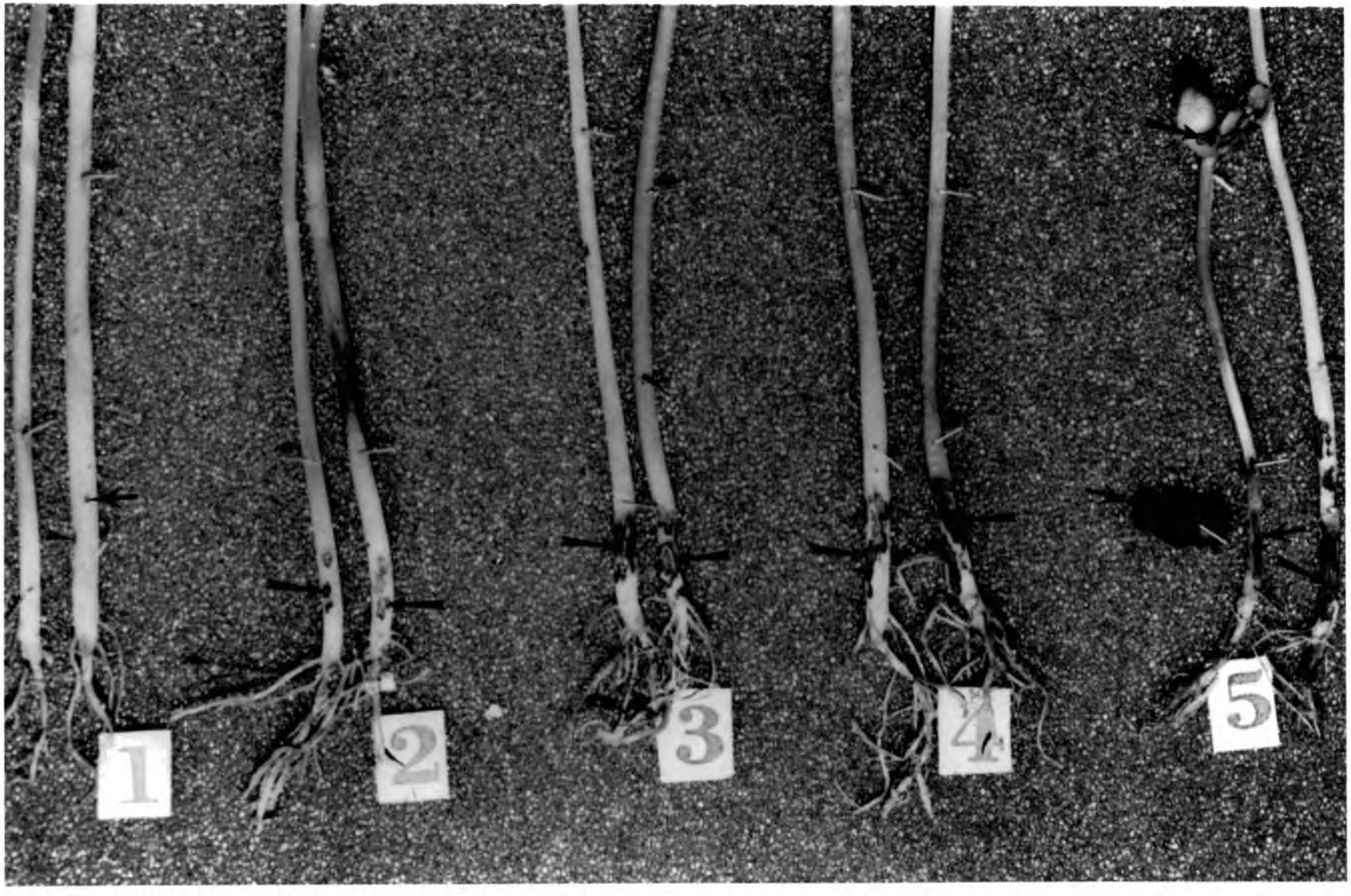
Five isolates of R. solani isolated from five different host were tested for virulence on bean. Four isolates were obtained from Dr. W. H. Ko at the Hawaii Agriculture Experiment Station at Hilo. These were isolates from Anthurium (Anthurium andraeanum), potato (Solanum tuberosum), silkwood (Flindersia brayleyana), and trefoil (Lotus corniculatas). The other isolate was obtained from infected hypocotyls of bean plants from Poamoho Agriculture Experiment Station.

Bean lines PI 165426 and Harvester were used in this test because of their previously reported response to R. solani infection (McLean et al., 1968; Prasad and Weigle, 1970; Deaken and Dukes, 1975). Two tests were conducted, one in June 1978, and the other in December 1978. Each test included two replicates of the two bean lines, five isolates, and a control which received no inoculum. Each treatment included 10 seeds.

Host Materials

Thirteen possibly resistant and four probably susceptible bean lines (Table 1) were tested. PI 165426, Venezuela 54, PI 181954, and PI 318700 were lines reported to be highly resistant to R. solani by McLean et al. (1968). PI 165426 was also reported to be resistant to

Figure 1. Infected bean hypocotyls classified into the disease classes 1 to 5. Lesions are indicated by arrows.


1
2
3
4
5

R. solani by several other workers such as Prasad and Weigle (1970), and Deaken and Dukes (1975). It has also been reported resistant to several other soil diseases like root knot nematodes (Fassuliotis et al., 1970) and Fusarium solani f. phaseoli (Smith and Housten, 1960). Cornell 2114-12 was used by Dickson and Boettger (1977) as the R. solani resistant parent for their studies on breeding multiple root rot resistance in snap beans. It has also been reported resistant to F. solani f. phaseoli, Pythium ultimum and Thielaviopsis basicola (Hassan et al., 1971; Dickson and Boettger, 1977). PI 226895 and PI 109895 were some of the additional sources of resistant bean lines reported by Prasad and Weigle (1970). B 4096, Wisconsin RRR77 and RRR83 are the only three breeding lines released as resistant to R. solani. These lines have desirable horticultural characteristics. PI 203958 R-275 has been reported as a good multiple root rot resistant line (Bravo et al., 1969). It has been widely used as a resistant parent for F. solani f. phaseoli, T. basicola, and P. ultimum (Hassan et al., 1971; Boomstra et al., 1977). Breeding line 69-3-21-13 was derived from a cross between PI 203958 R-275 and Manoa Wonder (Hartmann, personal communication). It has not been tested specifically for R. solani disease. Cultivars Harvester and Contender have been used as standard susceptible checks in R. solani studies (Prasad and Weigle, 1970; Deaken and Dukes, 1975). Manoa Wonder and Hawaiian Wonder are two cultivars released by the University of Hawaii. Manoa Wonder is root knot nematode resistant (Hartmann, 1968) and is a widely used cultivar in Hawaii.

Table 1. Characteristics of *P. vulgaris* lines tested for *R. solani* infection

Line	Growth habit	Flower color	Seed color	Origin	*R. solani* reaction reported	Reference
PI 165426	vine	white	brown	Mexico	resistant	McLean *et al.*, 1968
Venezuela 54	bush	purple	black	Venezuela	"	"
PI 181954	bush	white	brown	Syria	"	"
PI 318696	vine	purple	brown	Mexico	"	"
PI 318700	bush	purple	brown	Mexico	"	"
Cornell 2114-12	vine	purple	brown	New York	"	Dickson and Boettger, 1977
PI 226895	vine	purple	black	France	"	Prasad and Weigle, 1970
PI 109859	bush	white	red	Venezuela	"	"
B 4096	bush	purple	purple	S. Carolina	"	Deaken and Dukes, 1975
Wisconsin RRR 77	bush	white	white	Wisconsin	"	Hagedorn and Rand, 1977
Wisconsin RRR 83	bush	white	white	Wisconsin	"	"
PI 203958 R-275	vine	purple	black	Idaho	"	Bravo *et al.*, 1969
Breeding line 69-3-21-13	vine	purple	black	Hawaii	not tested	-
Harvester	bush	white	white	U.S.A.	susceptible	Prasad and Weigle, 1970
Contender	bush	purple	brown	U.S.A.	"	Deaken and Dukes, 1975
Manoa Wonder	vine	lavender	brown	Hawaii	not tested	-
Hawaiian Wonder	vine	lavender	brown	Hawaii	"	-

Inheritance Studies

Four resistant and three susceptible lines were chosen and crosses were made between the resistant and susceptible parents and among the resistant parents. Reciprocal crosses were also made. F_1 seeds were tested for their reactions to R. solani. F_2 seeds were obtained by growing F_1 plants in the field. F_2 seeds were saved from each individual F_1 plant and tested separately. F_2 tests for each cross were repeated at least 4 times. All tests included as susceptible checks, Harvester or Hawaiian Wonder. Progeny testing in the F_2 and in F_3 generations was done by obtaining F_3 and F_4 seeds from individuals of known disease reaction which had been transplanted to the field after being tested in the greehnouse.

F_2 data from individual F_1 plants including reciprocals and the pooled data were tested by Chi-square goodness of fit test for their fit to various genetic ratios. Data from parental, F_1 and F_2 generations were used to estimate the broad sense heritability using the formula, $H = V_G / V_E + V_G$. Genetic variance (V_G) was estimated by the difference between the total variance (V_{F_2}) and the environmental variance (V_E). The environmental variance was estimated by the mean of the variances of the non-segregating generations, P_1, P_2, and the F_1.

A parent-offspring regression using the mean scores of the F_3 progenies on the scores of their F_2 parental plants was used to estimate the narrow sense heritability. Gene action was further evaluated from the distribution of parental, F_1, F_2, F_3, and F_4 mean scores.

RESULTS AND DISCUSSION

Pathogen Variability and Virulence

The results of the comparative virulence test with five isolates are presented in Table 2. With the resistant bean line PI 165426, only small differences between the isolates were noted with only a few plants infected by any of the isolates. These differences were not significant. The few plants that were infected had small to medium brown surface lesions, corresponding to disease class 2. With the susceptible bean line, Harvester, however, significant differences between isolates were observed with the isolates from potato and anthurium being less virulent than the other three. Harvester plants were infected severely by the isolates from bean, trefoil, and silkwood, with symptoms corresponding to disease classes 3, 4, and 5. All 5 isolates behaved similarly on the two testing dates. Since the isolate from bean was the most pathogenic on Harvester but was not pathogenic on PI 165426, it was used in all subsequent tests in this study.

Response of Bean Hosts

The results of screening the 17 bean lines to the Poamoho bean isolate are presented in Table 3. The means of the resistant lines ranged from 1.08 to 2.10 and the susceptible lines from 3.93 to 4.88. The first 5 resistant lines were highly resistant having most plants in class 1, a few in class 2, and none in class 3. The other 8 resistant lines had more individuals in class 2 and some had a few in

Table 2. Comparative virulence of the 5 different isolates of R. solani inoculated on Resistant (PI 165426) and Susceptible (Harvester) snap bean

Line	Isolate	Number of Infected Plants[z] June 1978		Dec. 1978		Total	Mean[y]
		Rep 1	Rep 2	Rep 1	Rep 2		
PI 165426	bean	1	0	2	1	4	1.0
	trefoil	2	1	1	1	5	1.3
	silkwood	1	1	2	3	7	1.8
	anthurium	1	2	1	0	4	1.0
	potato	0	0	1	1	2	0.5
	control	0	0	0	0	0	0.0
Harvester	bean	10	10	9	8	37	9.3[a]
	trefoil	9	9	8	7	33	8.3[ab]
	silkwood	9	8	7	9	33	8.3[ab]
	anthurium	8	7	8	7	30	7.5[b]
	potato	2	4	5	2	13	3.3[c]
	control	0	0	0	0	0	0.0

[z] Plants classified in disease classes 2,3,4, and 5
[y] Means of isolates tested on Harvester followed by the same letter are not significantly different. BLSD at 5% level = 1.3.

Table 3. Classification of snap bean lines for R. solani infection

Line	Infection Class					Total # of Plants	Disease Rating	
	1	2	3	4	5		Mean[z]	Variance
Cornell 2114-12	36	3				39	1.08[a]	0.10
PI 226895	26	4				30	1.13[a]	0.12
PI 165426	33	6				39	1.15[a]	0.13
Venezuela 54	13	6				19	1.32[a]	0.23
Breeding line 69-3-21-13	11	8				19	1.42[a]	0.32
PI 318696	9	9				18	1.50[a]	0.37
Wisconsin RRR 77	13	15	1			29	1.58[a]	0.32
B 4096	18	15	6			39	1.69[a]	0.53
PI 318700	11	26				37	1.70[a]	0.21
PI 203958 R-275	6	13	1			20	1.75[a]	0.20
Wisconsin RRR 83	7	11	2			20	1.75[a]	0.41
PI 109859	1	17	1			19	2.00[a]	0.11
PI 181954		18	2			20	2.10[a]	0.10
Manoa Wonder			9	25	6	40	3.93[b]	0.38
Contender			3	6	30	39	4.67[b]	0.37
Hawaiian Wonder			1	5	34	40	4.83[b]	0.20
Harvester				5	35	40	4.88[b]	0.11

[z]Means followed by the same letter are not significantly different BLSD at 5% level = 1.21.

class 3. The last 3 susceptible lines were highly susceptible having most plants in class 5 and a few in classes 4 and 3. Manoa Wonder plants were mostly in class 4, and therefore it was a little less susceptible than the other susceptible lines, although the difference was not significant. All lines exhibited low variances. There were no significant differences among the resistant lines but all resistant lines were significantly different from the susceptible lines.

On the basis of this test, the three most resistant lines, Cornell 2114-12, PI 226895, and PI 165426, and slightly less resistant B 4096 were selected as resistant parents for the inheritance study. B 4096 was included because of its desirable horicultural characteristics and because of the experience I had working with its originators at Charleston, South Carolina. The two most susceptible lines, Harvester and Hawaiian Wonder, were selected as the susceptible parents. Manoa Wonder was also included as a susceptible parent because it is a widely grown cultivar locally and was slightly less susceptible.

On the basis of their parentage, all crosses were classified into 5 different groups as follows:

Group 1 - The most susceptible parents (Harvester and Hawaiian Wonder) x the most resistant ones (Cornell 2114-12, PI 226895, and PI 165426).

Group 2 - The most susceptible parents (Harvester and Hawaiian Wonder) x the slightly less resistant B 4096.

Group 3 - The less susceptible Manoa Wonder x the most resistant parents (Cornell 2114-12, PI 226895, and PI 165426).

Group 4 - The less susceptible Manoa Wonder x the slightly less resistant B 4096.

Group 5 - Resistant x resistant parents.

F_1 Generation Results

The results of testing F_1 plants are presented in Table 4. The reciprocal means of each cross (Appendix Table 20) were tested by the Student's t-test and was found to show no significant differences. Although the numbers are small, differences can be noted between the different groups. The lowest means (1.17 to 1.63) were observed in Group 5 which include the crosses among the resistant parents. The next lowest means (1.95 to 2.25) were in Group 3, which includes the less susceptible Manoa Wonder crosses with the most resistant parents. Next is the Group 1 (2.83 to 2.94) which includes the crosses of the most susceptible and the most resistant parents. The highest F_1 means (3.05, 3.26, 3.28) were found in Groups 2 and 4, which include all the crosses of the slightly less resistant B 4096 with susceptible plants. The differences between the means were not tested statistically because such information would be of little use.

F_2 Generation Results

The results of testing the F_2 plants are presented in Table 5. The most obvious feature of these data is the greater segregation and higher variances in Groups 1 to 4. Group 5 still shows very low means

Table 4. Classification of F_1 Plants for *Rhizoctonia solani* infection (Reciprocal crosses combined[z])

Parents	Infection Class and Frequency 1	2	3	4	5	Total	Mean	Variance
Group 1								
Harvester x PI 165426		5	18	1		24	2.83	0.22
Harvester x PI 226895		4	15	2		21	2.90	0.29
Harvester x 2114-12		2	14	1		17	2.94	0.13
Haw. Wonder x PI 165426		3	14	2		19	2.93	0.27
Haw. Wonder x PI 226895		4	16	2		24	2.83	0.33
Haw. Wonder x 2114-12		4	14	1		19	2.85	0.25
Group 2								
Harvester x B 4096		3	11	9		23	3.26	0.47
Haw. Wonder x B 4096		3	9	9		21	3.28	0.53
Group 3								
Man. Wonder x PI 165426		13	3			16	2.18	0.16
Man. Wonder x PI 226895		18	6			24	2.25	0.20
Man. Wonder x 2114-12	3	15	2			20	1.95	0.26
Group 4								
Man. Wonder x B 4096		5	8	6		19	3.05	0.61
Group 5								
PI 165426 x B 4096	9	2	1			12	1.33	0.43
2114-12 x B 4096	12	6	3			21	1.57	0.57
PI 165426 x 2114-12	19	4				23	1.17	0.15
PI 226895 x 2114-12	8	3				11	1.20	0.22
PI 165426 x PI 226895	10	4				14	1.28	0.22
PI 226895 x B 4096	3	5	1			9	1.63	0.44

[z] See Appendix, Table 20 for individual F_1 data.

Table 5. Classification of F_2 plants for *R. solani* infection (Pooled Data[z])

Parents	Infection Class 1	2	3	4	5	Total # of Plants	Mean	Variance
Group 1								
Harvester x PI 165426	78	129	195	111	78	586	2.99	1.42
Harvester x PI 226895	40	91	139	66	47	383	2.98	1.31
Harvester x 2114-12	65	135	200	108	66	574	2.96	1.33
Haw. Wonder x PI 165426	64	178	245	134	66	687	2.97	1.21
Haw. Wonder x PI 226895	44	69	89	64	32	298	2.90	1.46
Haw. Wonder x 2114-12	42	94	145	89	63	433	3.08	1.38
Group 2								
Harvester x B 4096	27	87	177	105	57	453	3.17	1.13
Haw. Wonder x B 4096	16	80	207	75	52	430	3.15	0.97
Group 3								
Man. Wonder x PI 165426	160	277	134	73	11	656	2.23	0.99
Man. Wonder x PI 226895	131	216	115	57	13	532	2.25	1.04
Man. Wonder x 2114-12	109	255	123	56	12	555	2.29	0.93
Group 4								
Man. Wonder x B 4096	129	340	275	119	40	903	2.56	1.06
Group 5								
PI 165426 x B 4096	139	31	5	-	-	175	1.23	0.25
2114-12 x B 4096	89	45	6	-	-	140	1.41	0.32
PI 165426 x 2114-12	296	41	1	-	-	338	1.12	0.13
PI 226895 x 2114-12	76	13	1	-	-	90	1.16	0.16
PI 165426 x PI 226895	91	6	-	-	-	97	1.06	0.15
PI 226895 x B 4096	39	11	6	-	-	56	1.41	0.46

[z]See Appendix, Table 21 for individual F_2 family data.

Table 6. Analysis of Variance of the 12 crosses included in Groups 1 to 4

Source of Variation	df	ss	ms	F
Among Crosses	11	754.70	68.60	57.88**
Within Crosses	6478	7683.82	1.19	
Total	6489	8438.52		

**Significant at 1% level

Table 7. Comparisons among the F_2 means with a posteriori test using a critical sum of square[z]

Comparison	SS	Significance at 5%
6 means in group 1	7.82	NS
2 means in group 2	0.06	NS
3 means in group 3	1.14	NS
Between group 1 and 2 means	25.84	Significant
Between group 1 and 3 means	454.77	Significant
Between group 1 and 4 means	116.95	Significant
Between group 2 and 3 means	481.12	Significant
Between group 2 and 4 means	163.96	Significant
Between group 3 and 4 means	53.53	Significant

[z]Critical sum of square = $(a-1)\ MS_{within} \times F_{\alpha}[a-1, a(n-1)]$

$= 11 \times 1.19 \times 1.79 = 23.35.$

a = number of crosses compared.

Table 8.

Bartlett's homogeneity of variance test for the F_2 progenies

Variance Tested	Adjusted X^2* Statistic	Critical Value of $X^2[0.01, (n)]$	Significance	Nature of Variance
For all the 52 progenies in groups 1 to 4	156.17	77.39	Sig.	Heterog.
For the 25 individual progenies tested in group 1	37.45	44.31	NS	Homog.
For the 10 individual progenies tested in group 2	21.95	21.66	Sig.	Heterog.
For the 12 individual progenies tested in group 3	23.50	24.73	NS	Homog.
For the 5 individual progenies tested in group 4	14.91	13.28	Sig.	Heterog.

* $X^2 = 2.306 \left\{ \left[\sum_1^a (n-1) \right] \log \bar{s}^2 - \sum_1^a (n_i-1) \log s_i^2 \right\}$ $\left(\bar{S} = \sum^a (n-1) S_i / \sum^a (n-1) \right)$

Correction factor (cf) $= 1 + \frac{1}{3(a-1)} \left[\sum_1^a \frac{1}{(n-1)} - \frac{1}{\sum_1^a (n-1)} \right]$

Adjusted $X^2 = X^2/cf$

and low variances. Generally, the F_2 means are very similar to the F_1 means, except for the one cross in Group 4.

The F_2 means of the 12 crosses in Groups 1, 2, 3, and 4 were then analyzed statistically. A preliminary analysis of variance showed significant differences among the means of these 12 crosses (Table 6). The means of progenies between and within the groups were then compared with a posteriori test (Table 7). This test showed significant differences between all the different groups, but not within the groups.

A test for homogeneity of variance was conducted using Bartlett's test on the individual progenies included in the four groups (Appendix Table 21). As expected, variances were heterogeneous for all 52 progenies. The variances of groups 1 and 3 were homogeneous, but those of groups 2 and 4, however, were significantly heterogeneous (Table 8).

Thus, there are 4 groups of segregating progenies which are all different from each other. Two of the groups (1 and 3) are homogeneous, while the other two (2 and 4) are heterogeneous. The latter two have in common the parent B 4096, the slightly less resistant one. The four groups will now be discussed individually.

Description of Groups

Group 1 This group includes all the progenies resulting from crosses between the most susceptible and the most resistant parents. It is a homogeneous group consisting of 25 progenies of 6 crosses. An example of this group is illustrated in

Figure 2. The F_1 means in this group were all close to 3.00 with low variances. Most individuals were in class 3 with usually a few more in class 2 than in class 4. The F_2 generation segregated into all 5 classes in an almost symmetrical distribution with about 10% of the individuals in class 1. Again, there were usually a few more individuals in class 2 than class 4. The variances in the F_2 were higher than in the F_1.

Group 2 This group includes the progenies from crosses of the most susceptible parents and the slightly less resistant B 4096. It is a heterogeneous group consisting of 10 progenies of 2 crosses. No representative example is shown because of the heterogeneity and variability within the group. In the F_1, the means, 3.26 and 3.28 (Table 4), were the highest of any of the segregating groups. The variances of 0.47 and 0.53 were also higher than the variances in Group 1. In the F_2, the means were higher than those in Group 1, but the variances were a little lower. All 5 classes appeared, but the number of individuals in class 1 was only about 5% or half that of Group 1. The variability found in this group is illustrated in Figures 3 and 4, which show the distribution of the most resistant and most susceptible F_2 progenies, respectively.

Group 3 This group includes the progenies from crosses between the less susceptible Manoa Wonder and the most resistant parents.

It is a homogeneous group consisting of 12 progenies of 3 crosses. An example of this group is illustrated in Figure 5. The F_1 means in Group 3 (Table 4) were much lower than those in Groups 1 and 2. More individuals were in class 2 than class 3. The F_1 variances were low. The F_2 means in Group 3 were also lower than those in Groups 1 and 2 (Table 5), but the F_2 variances were similar to those in Group 2 which were lower than in Group 1. Although all 5 classes appeared in the F_2, the most frequent class was 2 rather than 3. There were few or no individuals in class 5. About 25% of the population was in class 1, much higher than Groups 1 and 2.

Group 4 This group includes the progenies from crosses between the less susceptible Manoa Wonder and the slightly less resistant B 4096. It is a heterogeneous group consisting of 5 progenies of 1 cross. The F_1 mean and variance were high, similar to those found in Group 2. In the F_2, the mean was lower than in Group 2, with the highest number of individuals in class 2. The variance in the F_2 was low like in Groups 2 and 3. Two extreme examples of this group are illustrated in Figures 6 and 7.

Group 5 This group includes the progenies from crosses among the resistant lines. It is a homogeneous group consisting of 11 progenies of 6 crosses. An example of this group is illustrated in Figure 8. The F_1 and F_2 means in this group were

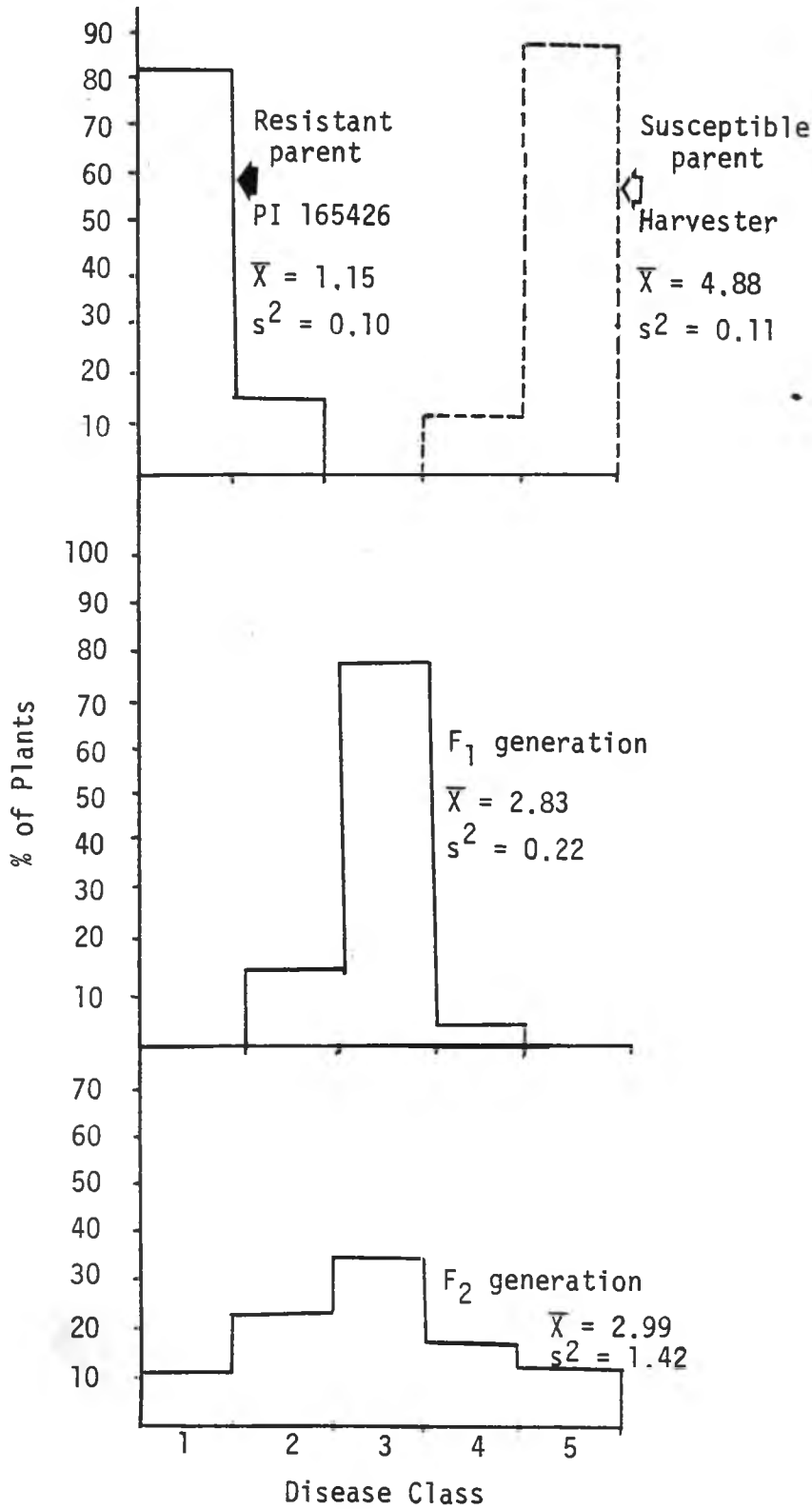


Figure 2. A Group 1 cross (Harvester x PI 165426)
$\overline{X}$ = Mean, s^2 = Variance

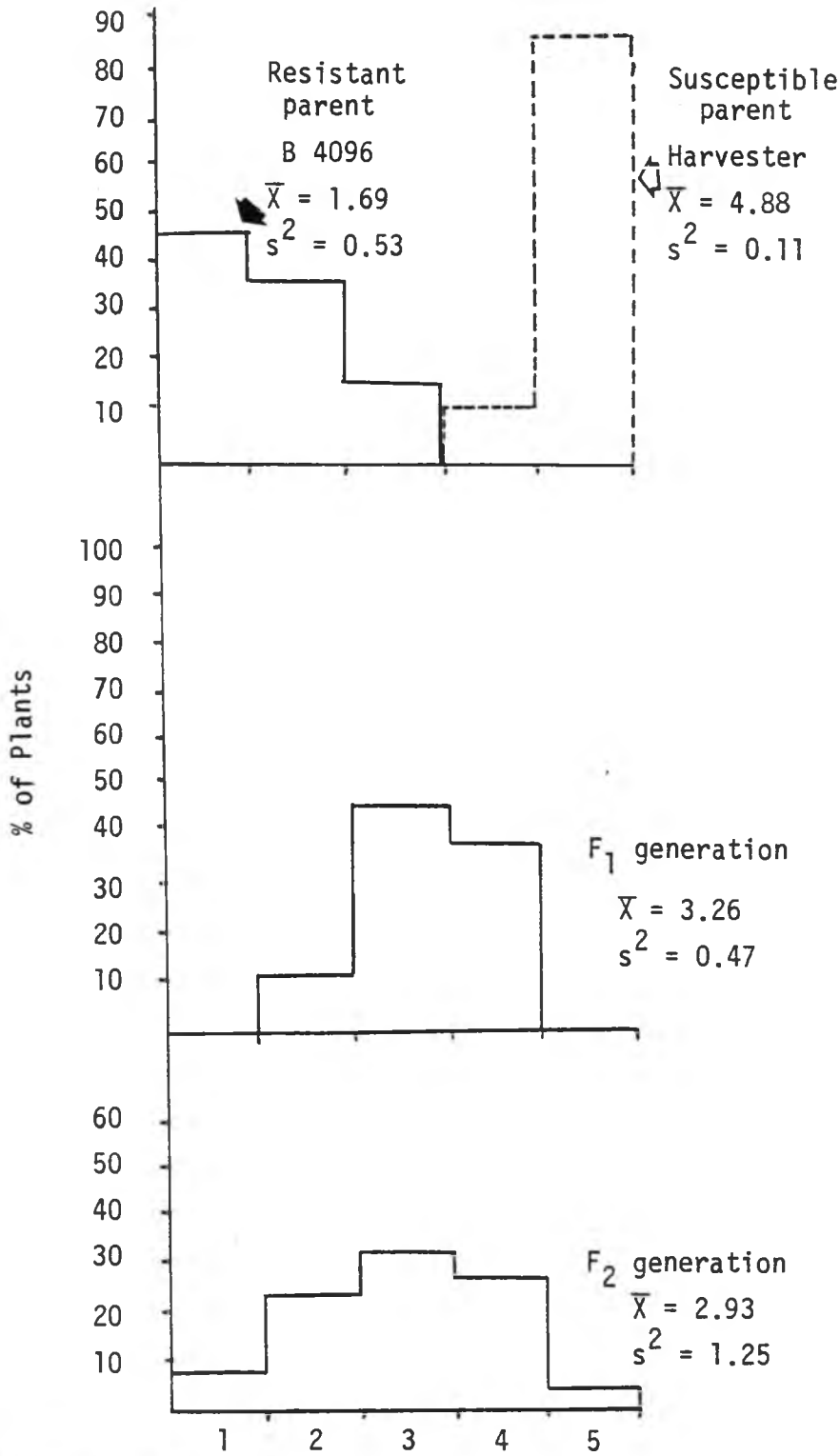


Figure 3. A Group 2 'resistant' F_2 segregation from the cross, Harvester x B 4096

$\overline{X}$ = Mean, s^2 = Variance

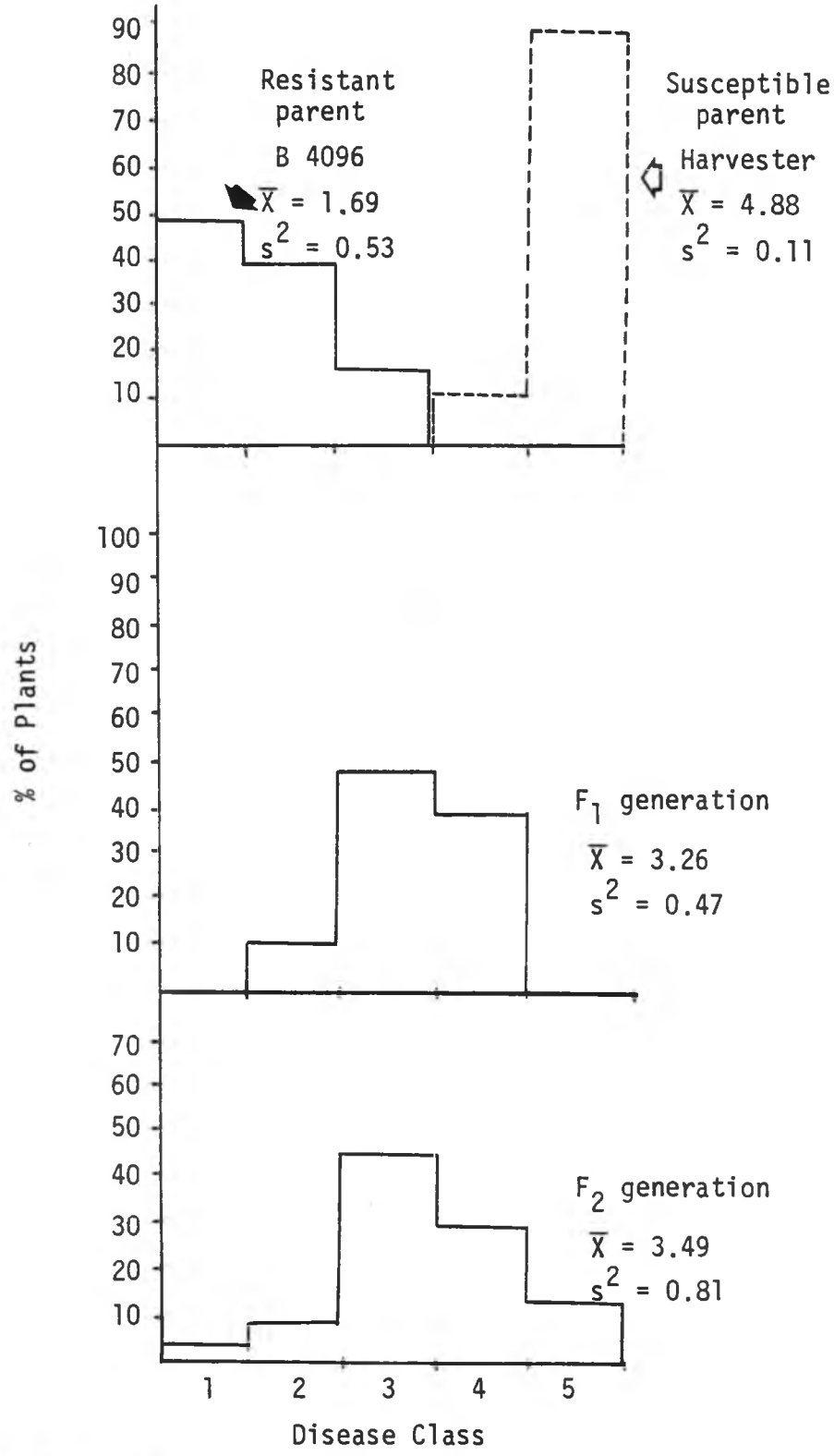


Figure 4. A Group 2 'susceptible' F_2 segregation from the cross, Harvester x B 4096

$\overline{X}$ = Mean, s^2 = Variance

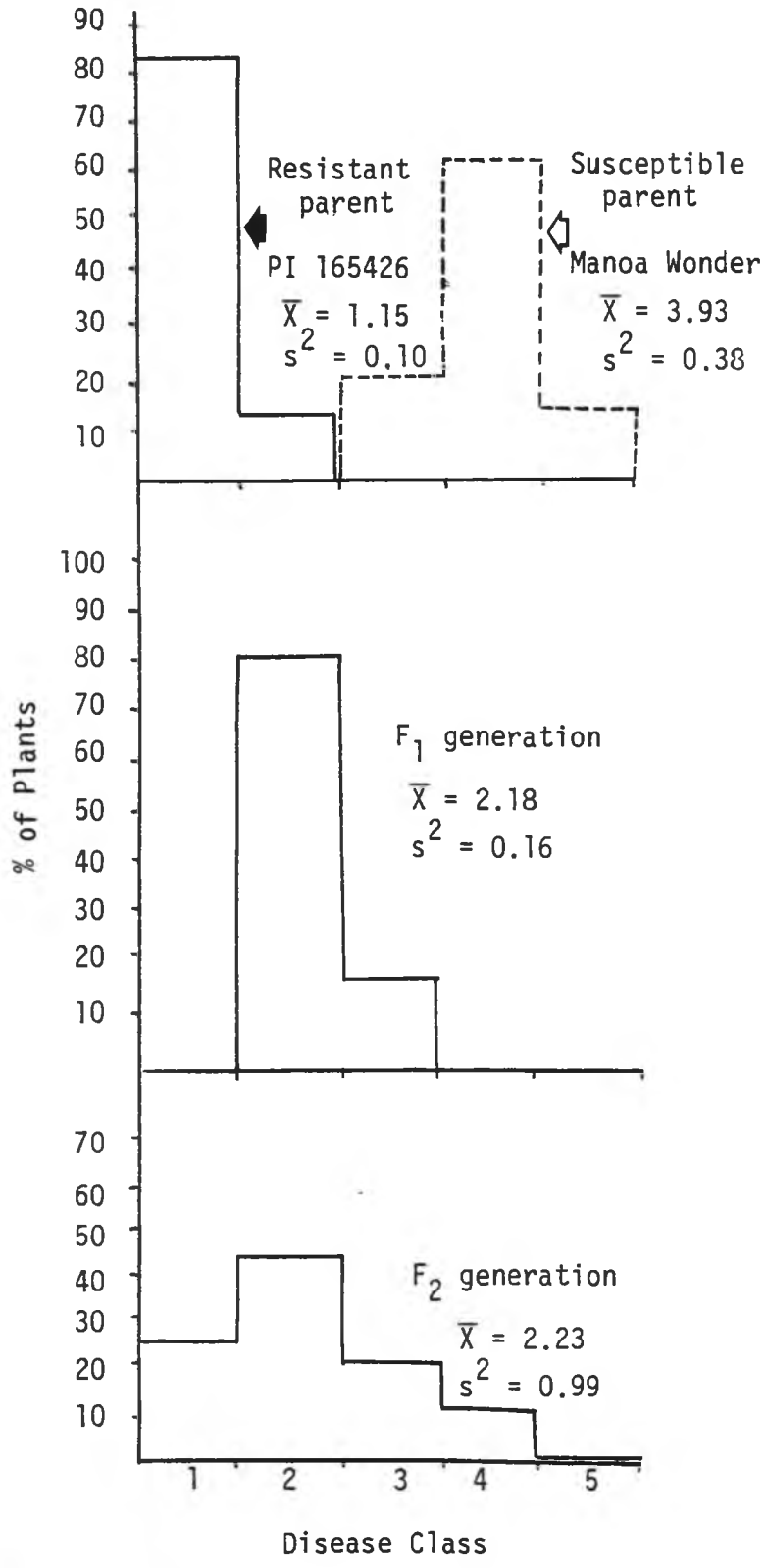


Figure 5. A Group 3 cross (Manoa Wonder x PI 165426
$\overline{X}$ = Mean, s^2 = Variance

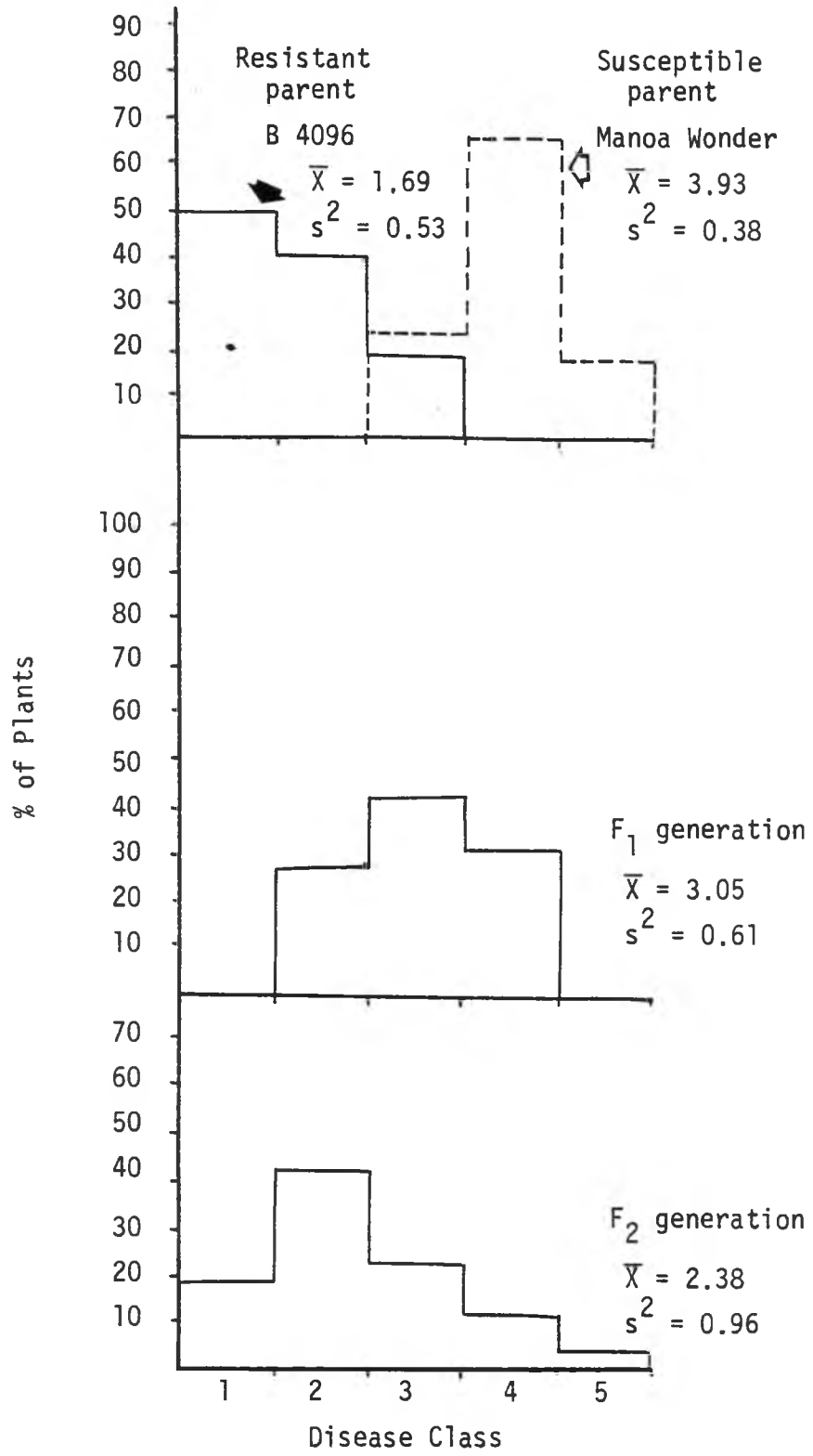


Figure 6. A Group 4 'resistant' F_2 segregation (Manoa Wonder x B 4096)

$\overline{X}$ = Mean, s^2 = Variance

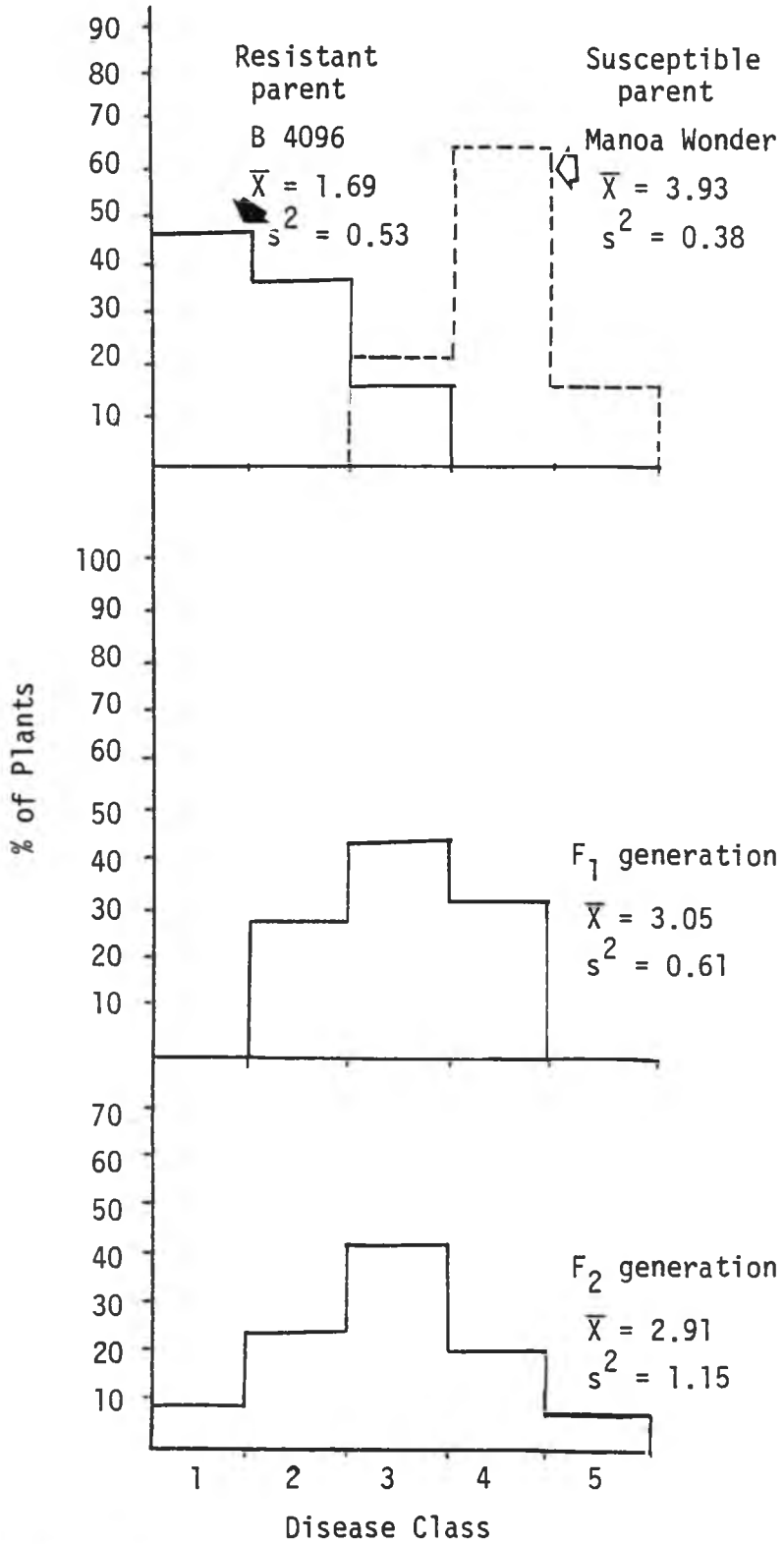


Figure 7. A Group 4 'susceptible' F_2 segregation (Manoa Wonder x B 4096)
$\overline{X}$ = Mean, s^2 = Variance

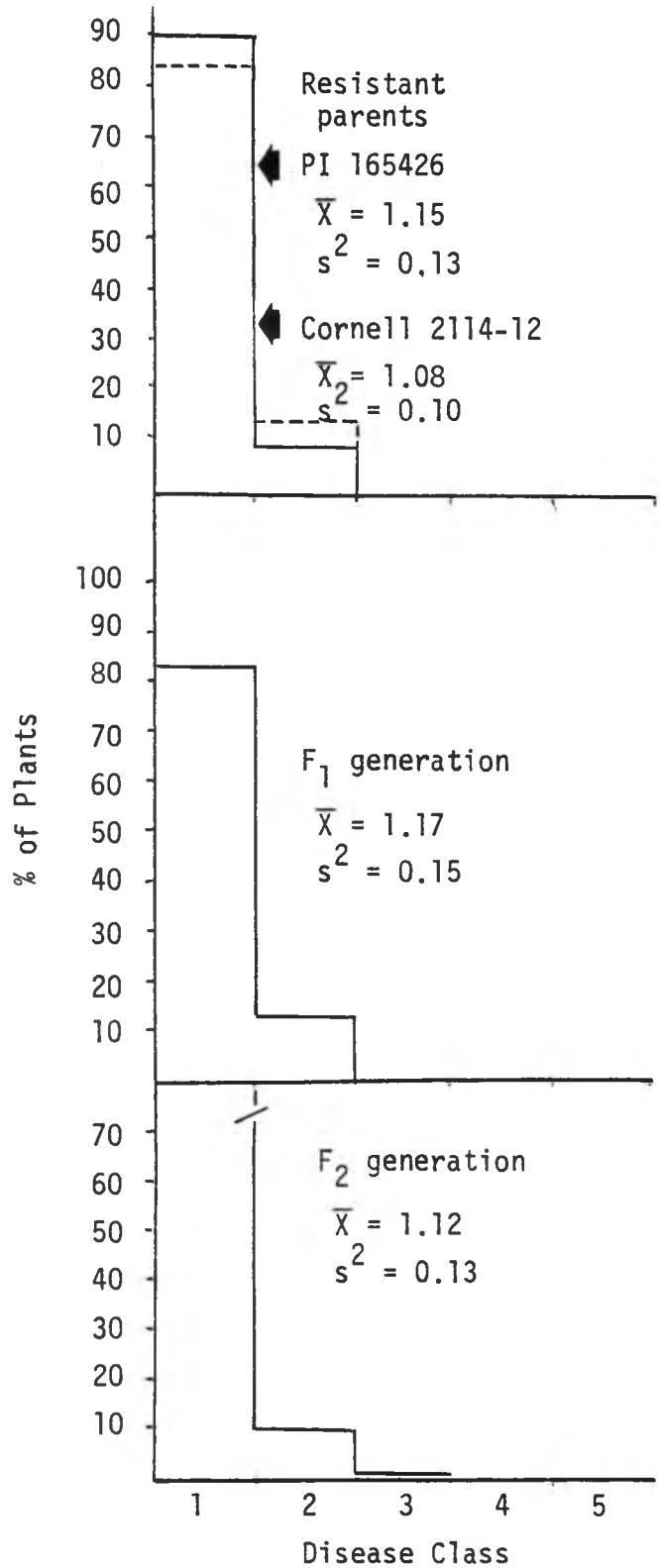


Figure 8. A Group 5 cross (PI 165426 x Cornell 2114-12)
$\bar{X}$ = Mean, s^2 = Variance

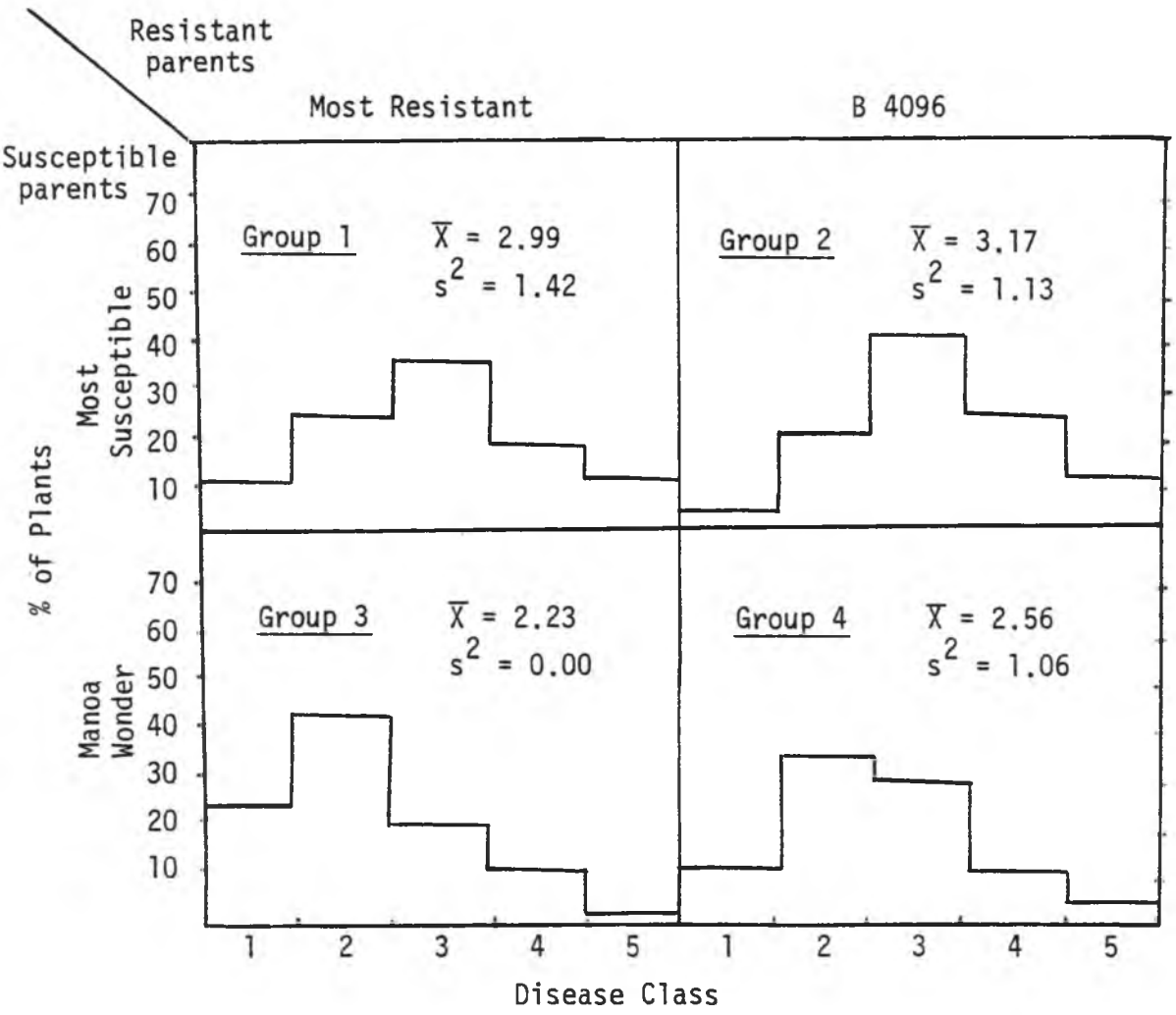


Figure 9. F_2 segregation in Groups 1, 2, 3, and 4 (pooled data of one cross in each Group)

$\overline{X}$ = Mean, s^2 = Variance

Group 1 - Harvester x PI 165426
Group 2 - Harvester x B 4096
Group 3 - Man. Wonder x PI 165426
Group 4 - Man. Wonder x B 4096

very low, with most individuals in class 1 and only very few in class 3 (Tables 4 and 5). Variances in both the F_1 and F_2 were also low. However, when B 4096 was one of the parents, the variances in both the F_1 and F_2 were higher than when it was not involved.

The F_2 results observed are summarized in Figure 9. It can be seen that the most resistant parents (Cornell 2114, PI 226895, and PI 165426) convey more resistance to their F_2 progeny (Groups 1 and 3) than B 4096 does (Groups 2 and 4) and also that the slightly less susceptible Manoa Wonder also conveys some resistance to its F_2 progeny (Groups 3 and 4).

Genetic Ratios Observed in Group 1

In Group 1, the F_2 distribution showed an apparently normal distribution with all five classes represented and about equal numbers in classes 1 and 5 as well as classes 2 and 4, with the highest number of individuals in class 3 (Figure 2). The symmetry of the distribution as well as the intermediate action of the F_1 imply that there are quantitative genes involved with additive gene action. However, the presence of about 10% of the individuals in each of the extreme classes also indicates that the number of genes involved is small, since with 2 pairs of genes segregating in a 1:4:6:4:1 ratio, there should be only about 6.25% in each of the extreme classes. The Chi-square values, however, showed a poor fit to a 1:4:6:4:1 ratio (Table 9). An attempt was next made to fit a 3 factor 1:6:15:20:15:6:1 ratio with 6 or 5 genes for resistance falling in class 1, 4 resistant

Table 9. Testing F_2 segregation ratios for Group 1 (Most Susceptible x Most Resistant) crosses[z]

Cross	Infection Class and Frequency					Chi-square			
	1	2	3	4	5	1:4:6:4:1 ratio	7:15:20:15:7 ratio	5:11[y] ratio	22:42[x] ratio
Group 1									
Harvester x PI 165426	73	129	195	111	78	94.32**	10.51*	2.87	0.30
Harvester x PI 226895	40	91	139	66	47	42.51**	10.10*	1.24	0.46
Harvester x 2114-12	65	135	200	108	66	37.48**	6.56	0.83	0.38
Haw. Wonder x PI 165426	64	178	245	134	66	31.81**	13.20*	5.14*	0.23
Haw. Wonder x PI 226895	44	69	89	64	32	48.60**	4.39	6.70**	1.21
Haw. Wonder x 2114-12	42	94	145	89	63	63.27**	8.63	0.71	1.54

[z] See Appendix, Table 22 for individual progenies

[y] 1:4:6:4:1 segregation with at least 2 genes necessary for susceptibility

[x] 1:6:15:20:15:6:1 segregation with at least 3 genes necessary for susceptibility

* Significant at 5% level
** Significant at 1% level

genes in class 2, 3 resistant genes in class 3, 2 resistant genes in class 4, and 1 or 0 genes in class 5, giving a ratio of 7:15:20:15:7. This combination would cause about 10% of the population to fall into the extreme classes 1 and 5. The fit to this ratio was better (Table 9). A 4 factor 1:8:28:56:70:56:28:8:1 ratio was not considered because no combination of numbers will produce 5 classes with about 10% of the population in the extreme classes.

The F_2 data were then tested further by combining classes 1 and 2 as resistant and classes 3, 4, and 5 as susceptible to test against 5:11 (2 genes) and 22:42 (3 genes) ratios (Table 9). Tested this way, 4 out of 6 combined populations and 20 out of 25 individual progenies (Appendix Table 22) fit a 5:11 ratio, but all the combined and individual progenies fit a 22:42 ratio.

Genetic Ratios Observed in Group 3

In Group 3, which was also homogeneous like Group 1, the segregation pattern was quite different from Group 1 with more plants in the resistant classes 1 and 2 than in the susceptible classes 3, 4, and 5 in the F_2 generation. If it is assumed that Manoa Wonder has more individuals in class 4 than Hawaiian Wonder or Harvester (Table 3) because it has some genes for resistance, then its progeny would also be expected to be more resistant, as they are. Since the Group 1 crosses best fit a ratio which assumes 3 pairs of segregating genes, the Group 3 crosses might be expected to fit a ratio which assumes only 2 pairs segregating while the other pair is homozygous for resistance. The 3 combined populations (Table 10) and 12 individual

Table 10. Testing F_2 segregation ratio of Group 3 (most resistant x less susceptible) crosses[z]

Parents	Infection Class and Frequency					Total	Chi-square 11:5 ratio
	1	2	3	4	5		
Man. Wonder x PI 165426	160	277	134	73	11	656	1.20
Man. Wonder x PI 226895	131	216	115	57	13	532	2.94
Man. Wonder x 2114-12	109	255	123	56	12	555	2.72

[z]See Appendix, Table 23 for individual progenies.

progenies (Appendix Table 23) all fit such a 2 factor, 11 resistant : 5 susceptible ratio.

Genetic Ratios in Groups 2, 4, and 5

The crosses included in Groups 2 and 4, all of which involved the resistant parent, B 4096, were not homogeneous and therefore not combined and tested to fit any ratios. Individual progenies sometimes fit the ratios found with the other resistant parents, but often did not (Appendix Table 24). In Group 2, 6 of the 10 progenies fit a 22:42 ratio (and even a 5:11 ratio) as found for Group 1, but 4 progenies did not. Likewise, in Group 4, 2 of the 5 progenies fit a 5:11 ratio like in Group 3, but the other 3 clearly did not. It is, therefore, concluded that B 4096 does not always transmit the same genes to its progeny.

In Group 5, which includes the crosses among the resistant parents, the F_1 and F_2 means were all low and comparable to the means of the parents. There seemed to be no segregation in the F_2, except possibly for a small amount in some crosses involving B 4096. One F_2 progeny of the cross between B 4096 and PI 165426 arising from one F_1 seed had 89 individuals in class 1, 21 in class 2, and 2 in class 3 (Appendix Table 21). If the 2 individuals in class 3 are considered to be errors in classification, then the 89:21 ratio gives an acceptable fit to a single gene 3:1 ratio with a chi-square of 1.76.

Prelimilary Conclusions

On the basis of the parental, F_1 and F_2 results, it is tentatively concluded, therefore, that Cornell 2114-12, PI 226895, and

PI 165426 all carry the same genes for resistance and differ from Harvester and Hawaiian Wonder by 3 pairs of genes which are equal and additive in action. It is also concluded that Manoa Wonder carries 1 pair of genes for resistance and thus differs by only 2 pairs from Cornell 2114-12, PI 226895, and PI 165426. Manoa Wonder most likely received these genes from its parent, Alabama #1 (Hartmann, personal communication) which has been reported to have some resistance (McLean et al., 1968).

B 4096 appears to still be segregating for resistance based on its production of non-homogeneous F_2 progenies, its slightly higher variance as a parent, and some segregation when it is crossed with other resistant parents. It has been observed to segregate for flower and seed color also.

Progeny Testing

In order to test the accuracy of the individual plant classifications in the F_2 generation, 162 plants representing all five classes were progeny tested (Tables 11-15). The first digit of each plant number indicates the group from which it came, the second digit indicates the class of the F_2 parents, and the remaining digits are the plant number. Plants were selected mostly from crosses in Groups 1 to 4. Class 5 plants tested were limited to those which put out new roots from above the hypocotyl lesion areas and thus survived the severe infection level. Therefore, class 5 progeny testing may be biased by testing only the surviving plants.

In class 1 (Table 11), 31 plants were tested. 3 plants (118,

215, 414) produced individuals in classes 3 and 4, while 2 plants (111, 312) produced a few individuals in class 3. These plants probably should have been in class 3. The other 26 plants produced progenies with means ranging from 1.11 to 1.79 and seem to have been correctly classified. In class 2 (Table 12), out of the 41 plants tested, 8 plants (129, 1211, 222, 223, 325, 423, 425) segregated widely with some individuals in class 5 and probably should have been in class 3. The remaining 33 seem correctly classified. In class 3 (Table 13), out of the 40 plants tested, low values for 4 progenies (138, 321, 338, 530) indicate that their F_2 parents may have been classified too high. Plant 530, especially, was one of the few class 3 plants from Group 5 (resistant x resistant) and apparently is really in class 2. One plant (132) may have been an escape in the F_2. In class 4 (Table 14), of the 32 plants tested, 5 plants (143, 1411, 246, 440, 445) probably should have been in class 3. In class 5 (Table 15) also, a few plants (250, 350, 351, 452) probably were classified too high and should have been in class 4. Two such plants (350, 351) were from Group 3 parents which had very few or no individuals segregating into class 5.

The evaluation of the F_3 progenies, therefore, indicates little misclassification in the F_2 generation. Misclassifications that were made in the F_2 only resulted in placing a plant in the class adjacent to its true class. This kind of misclassification is not unexpected when a continuous distribution is artificially divided into discrete classes.

Table 11. Classification of F_3 progenies of class 1 F_2 plants for R. solani infection

Plant No.	F_3 Mean	Infection Class and Frequency				
		1	2	3	4	5
110[z]	1.11	69	9			
111	1.71	19	34	3		
112[z]	1.12	81	12			
113	1.31	66	30			
114	1.26	42	13			
115[z]	1.19	52	12			
116	1.32	46	22			
117	1.79	9	22			
118	2.28	16	13	12	9	
119	1.31	41	19			
1110	1.30	61	23			
1111[z]	1.20	72	18			
1112	1.72	30	57			
211	1.20	51	13			
212	1.26	28	10			
213	1.52	24	20			
214	1.36	41	18			
215	2.67	2	12	14	5	
311	1.42	35	25			
312	1.91	10	29	6		
313	1.36	35	20			
314	1.31	51	18			
315[z]	1.20	64	16			
316	1.58	17	23			
317	1.45	30	25			
318	1.29	52	22			
411	1.24	68	22			
412	1.27	50	18			
413	1.57	32	43			
414	2.52	4	23	15	7	
415	1.29	36	15			

[z] Probable homozygous families

Table 12. Classification of F_3 progenies of class 2 F_2 plants for *R. solani* infection

Plant No.	F_3 Mean	Infection Class and Frequency				
		1	2	3	4	5
120[z]	1.94	5	42	2		
121	2.33	10	18	21	3	
122	2.07	19	19	11	6	
123	1.98	20	21	9	5	
124[z]	2.00	6	48	2		
125	2.43	9	14	17	6	
126	2.27	13	23	12	7	
127	2.31	16	24	19	9	
128	2.34	13	21	10	7	
129	3.26	5	12	18	16	7
1210	2.62	6	26	21	13	
1211	2.71	12	42	20	18	9
1212	2.40	11	23	24	6	
1213	1.89	23	49	12	4	
1214	2.36		33	6	5	
220	2.19	2	22	8	1	
221	2.39	13	13	14	9	
222	2.78		24	8	7	3
223	2.57	8	11	11	3	4
224	2.22	14	22	15	6	
225	3.16		9	11	8	3
320[z]	1.90	27	63	15		
321	1.86	16	32	8		
322	2.48	14	18	19	9	
323	2.61	3	21	11	10	
324	2.21	27	40	13	13	
325	3.10	1	9	15	12	3
326	2.37	12	21	13	10	
327	2.53	8	43	16	19	
328[z]	1.96	7	37	5		
329	2.57	16	27	32	19	
3210	2.25	12	21	13	6	
3211	3.24		2	15	8	
420	2.44	17	22	14	13	
421	2.58		30	11	9	
422	2.31	13	20	16	7	
423	2.93	9	20	25	21	6
424	2.28	14	19	9	10	
425	2.76	13	26	14	19	7
426[z]	2.07	2	49	6		
427	3.04	3	21	30	27	
520[z]	1.86	6	19	2		

[z] Probable homozygous families

Table 13. Classification of F_3 progenies of class 3 F_2 plants for R. solani infection

Plant No.	F_3 Mean	Infection Class and Frequency				
		1	2	3	4	5
130	3.53		2	30	16	8
131	3.12	10	18	24	20	14
132	4.00		3	7	21	13
133	2.86	6	16	11	13	4
134	3.00		10	10	8	1
135	3.15	7	13	20	21	8
136	3.39		9	30	26	6
137	3.24	4	9	12	14	7
138	2.50	10	18	30	6	
139	3.06	6	8	19	11	6
1310	3.05		16	46	8	6
1311	3.37		1	19	14	9
1312	2.90	3	20	16	11	5
1313	2.93		19	17	10	3
230	3.20	1	6	12	8	3
231	2.52	9	15	12	7	2
232	3.20	5	13	11	9	4
233	2.85	3	11	9	13	1
234	3.04	2	6	26	12	
235	3.16	4	13	14	16	7
236	3.00		11	19	9	1
330	2.91	6	13	19	14	3
331	3.16		12	39	24	
332	2.84	7	13	10	8	6
333	2.81	8	13	17	11	4
334	2.85	9	15	13	13	6
335	3.22		4	39	12	2
336	3.09	3	16	19	20	4
337	3.00	6	14	19	22	2
338	2.63	9	30	26	18	
339	3.07		16	44	22	
3310	3.10		12	16	10	3
3311	2.80	10	13	23	9	6
430	3.10		10	15	14	
431	3.03	4	16	28	20	
432	2.84	6	21	18	17	3
433	2.89		14	34	8	
434	3.11		16	13	11	5
435	3.15	6	10	22	15	8
530[z]	2.19	1	21	6		

[z] Probable homozygous family

Table 14. Classification of F_3 progenies of class 4 F_2 plants for R. solani infection

Plant No.	F_3 Mean	Infection Class and Frequency				
		1	2	3	4	5
140	4.19			9	11	16
141	4.23			2	19	9
142	3.91		6	12	23	19
143	3.06	3	6	18	10	2
144	4.17		2	12	20	26
145	4.13		3	8	16	20
146[z]	4.07			5	28	8
147	3.44		10	19	24	6
148	3.38	3	6	12	17	5
149	3.79		7	16	16	18
1410[z]	4.02			5	33	6
1411	2.26	12	16	7	8	
240[z]	4.03			4	19	5
241	4.19		3	5	16	9
242	4.10		2	7	10	14
243	3.20	5	9	22	20	6
244	3.71	1	5	6	14	9
245[z]	4.06			4	21	6
246	3.06	2	12	14	13	3
340	3.53		5	13	15	7
341[z]	3.81			7	29	1
342	3.68		8	26	24	18
343	3.79		10	16	20	22
344	3.59			19	17	3
345	3.26	2	8	16	13	5
346	3.80			14	19	4
440	3.07	4	12	14	20	2
441[z]	4.08			1	19	3
442	3.79		6	9	23	9
443	4.00		6	14	20	26
444	3.30	2	7	12	10	6
445	2.97		18	32	16	

[z] Probable homozygous families

Table 15. Classification of F_3 progenies of class 5 F_2 plants for *R. solani* infection

Plant No.	F_3 mean	Infection Class and Frequency				
		1	2	3	4	5
150[z]	4.77				5	17
151	4.67				14	28
152	4.37				30	18
153	4.29			4	11	12
154	4.60				10	17
155	4.26			10	8	20
156	4.51				9	12
157[z]	4.74				8	23
250	3.61			11	3	4
251	4.53				13	15
252	4.55				9	11
253	4.10				7	9
350	3.82			7	19	2
351	3.92			5	17	3
450	4.64				9	16
451	4.31			6	8	15
452	3.95			7	9	6

[z] Probable homozygous families

Homozygotes

Additional evidence for the type of inheritance may be obtained from the kind of homozygotes observed among the F_2 progeny. If genes for resistance are designated as R, then with 3 pairs of genes controlling this character, there should be 4 kinds of homozygotes, RRRRRR for all the resistant genes, which should be class 1, RRRRrr for 2 of the 3 pairs, which should be class 2, RRrrrr for 1 of the 3 pairs, which should be class 4, and rrrrrr for none of the resistance genes, which should be class 5, according to the original hypothesis. On the other hand, if only 2 pairs of genes are controlling the character, there should be only 3 kinds of homozygotes, RRRR, class 1, rrrr, class 5, and RRrr, probably class 3 (intermediate between resistant and susceptible). Thus, the F_3 progenies were examined for the presence of probable homozygotes.

In class 1 (Table 11), at least 5 progenies (110, 112, 115, 1111, 315) seem to be homozygous for the class 1 level of resistance. In class 2 (Table 12), 6 plants (120, 124, 320, 328, 426, 520) have most individuals in class 2 with only a very few individuals in classes 1 and 3 and are probably homozygous for class 2. In class 3 (Table 13), almost all progenies segregated into 4 or 5 classes. Those with only 3 classes (331, 339, 430, 433) all had substantial numbers in classes 2 and 4 and do not seem to be homozygous. Plant 530 does seem to be homozygous, but it has previously been noted that this should have been in class 2. In class 4 (Table 14), 6 families (146, 1410, 240, 245, 341, 441) have most progeny in class 4 with only a very few individuals in classes 3 and 5 and seem to be homozygous, but it

should be remembered that only the less severely infected class 5 F_2 plants were able to survive to be progeny tested.

Thus, there are families which appear to be homozygous in classes 1, 2, 4, and 5, but not in class 3. The presence of homozygotes in these classes would confirm the theory that 3 pairs of genes are involved, and eliminate the possibility that only 2 pairs are involved. Therefore, 1 class 1 family (110) and 2 class 2 families (120, 124) that appeared to be homozygous were compared in the F_4 generation with a class 2 family (127) that seemed to be segregating (Table 16). Each F_4 family originated from an F_3 plant classified as class 1 in family 110 and class 2 in families 120, 124, and 127.

Family 110 is clearly homozygous for class 1. Nearly all individuals in all 5 F_4 families are in class 1 with only a few individuals in class 2.

Families 120 and 124 also seem quite clearly to be homozygous, but for class 2. With one exception (120-6) all 17 F_4 families have a very large number of individuals in class 2 and only a very small number in classes 1 and/or 3.

Family 127, on the other hand, is clearly much more variable. All but 2 of the 8 F_4 families tested have individuals more or less evenly distributed over classes 1 to 4, clearly different than with families 120 and 124. Possibly family 127-4 is now homozygous for class 2, though.

Testing F_2 Segregation from Progeny Tested Data

Since the F_2 plants selected for progeny testing were selected at

Table 16. Distribution of disease classes in F_4 progenies of F_3 families, 110, 120, 124, and 127

Family No.	F_4 Mean	Infection class and Frequency				
		1	2	3	4	5
Non-segregating						
110-1	1.14	36	6			
110-2	1.20	31	8			
110-3	1.23	16	5			
110-4	1.13	27	4			
110-5	1.19	41	10			
120-1	2.13	3	49	11		
120-2	1.81	6	27	-		
120-3	2.10	4	37	10		
120-4	2.14	1	16	4		
120-5	1.93	1	14	-		
120-6	1.72	12	21	-		
120-7	2.08	3	16	5		
120-8	2.12	1	20	4		
120-9	1.78	7	18	-		
124-1	1.90	5	29	1		
124-2	2.06	3	34	6		
124-3	1.80	8	33	-		
124-4	2.10	-	39	4		
124-5	2.05	7	41	10		
124-6	2.11	4	32	9		
124-7	2.09	-	19	2		
124-8	1.85	4	15	1		
Segregating						
127-1	2.31	19	21	17	12	-
127-2	2.41	13	18	15	9	1
127-3	2.57	4	29	11	13	-
127-4	1.92	10	22	7	-	-
127-5	2.69	-	18	11	7	-
127-6	2.27	13	21	19	5	-
127-7	2.30	11	19	14	6	-
127-8	2.63	-	22	9	6	1

random among the individuals within each class, the F_2 frequencies of the homogeneous groups (1 and 3) were examined for their fit to what would be expected on the basis of the three-gene pair hypothesis. Each F_2 plant was put in its proper classification according to its observed F_3 segregation and a decision was made on which of the segregation classes it appeared to fall into.

With 3 pairs of genes segregating, as in Group 1, the F_2 generation would be expected to segregate as follows:

1/64 homozygous class 1 with 6 resistance genes

6/64 heterozygous class 1 with 5 resistance genes that segregates 3 class 1:1 class 2

3/64 homozygous class 2 with 4 resistance genes

12/64 heterozygous class 2 with 4 resistance genes that segregates 5 class 1:6 class 2:4 class 3:1 class 4

8/64 heterozygous class 3 with 3 resistance genes that segregates 7:15:20:15:7

12/64 heterozygous class 3 with 3 resistance genes that segregates 1 class 2:2 class 3:1 class 4

3/64 homozygous class 4 with 2 resistance genes

12/64 heterozygous class 4 with 2 resistance genes that segregates 1 class 2:4 class 3:6 class 4:5 class 5

6/64 heterozygous class 5 with 1 resistance gene that segregates 1 class 4:3 class 5

1/64 homozygous class 5 with 0 resistance gene

F_2 plants from Group 1 in the different disease classes which had been

progeny tested were then compared with the expected proportions (Table 17). As can be seen, the fit of the expected and observed proportions was very close in all classes, even though the numbers were quite small.

Similarly, with 2 pairs of genes segregating and 1 pair homozygous for resistance as in Group 3, the F_2 is expected to segregate:

1/16 homozygous class 1 with 6 resistance genes
4/16 heterozygous class 1 with 5 resistance genes that segregates 3 class 1:1 class 2
2/16 homozygous class 2 with 4 resistance genes
4/16 heterozygous class 2 with 4 resistance genes that segregates 5 class 1:6 class 2:4 class 3:1 class 4
4/16 heterozygous class 3 with 3 resistance genes that segregates 1 class 2:2 class 3:1 class 4
1/16 homozygous class 4 with 2 resistance genes

F_2 plants in the different disease classes which had been progeny tested were then compared with the expected proportions (Table 18). As with Group 1, Group 3 results very closely fit the expected proportion of plants in each disease class. A much closer agreement between the observed and expected number would be likely if larger samples of plants from the different disease classes were progeny tested. Thus, the proportions of F_2 types observed are in complete agreement with the hypothesis that 3 gene pairs in Group 1 and 2 gene pairs in Group 3 are segregating for resistance.

Table 17. Testing of Group 1 segregation with progeny tested data

Type	Number of Plants		Chi-square
	Expected	Observed	
Class 1			
Homozygotes	1.85	4	4.42*
Heterozygotes-segregate	11.15	7	
Class 2			
Homozygotes	3.80	3	0.22
Heterozygotes-segregate	15.20	16	
Class 3			
Homozygotes	0.00	0	
Heterozygotes-segregate 7:15:20:15:7	5.20	6	0.21
Heterozygotes-segregate 1:2:1	7.80	7	
Class 4			
Homozygotes	2.61	3	0.08
Heterozygotes-segregate	10.40	10	
Class 5			
Homozygotes	0.86	2	1.76
Heterozygotes-segregate	5.14	4	

* Significant at 5% level

Table 18. Testing of Group 3 segregation with progeny tested data

Type	Number of Plants		Chi-square
	Expected	Observed	
Class 1			
Homozygotes	1.40	1	1.42
Heterozygotes-segregate	5.60	6	
Class 2			
Homozygotes	4.33	4	0.04
Heterozygotes-segregate	8.66	9	
Class 3			
Homozygotes	0.00	0	0.00
Heterozygotes-segregate	12.00	12	
Class 4			
Homozygotes	5.00	4	-
Heterozygotes-segregate	0.00	2	
Class 5			
Homozygotes	0.00	0	-
Heterozygotes-segregate	0.00	0	

Heritability Estimates

Broad sense heritability in F_2

The estimates of broad sense heritability which were calculated for the F_2 populations using the variances of the parental and F_1 populations to estimate the environmental variance, are given in Table 19. The heritability estimates for the Group 1 populations are high ranging from 83.5 to 91.4%, which indicates that most of the variability in these populations is due to genetic factors, as should be the case under these conditions of artificial inoculation. It has already been shown that relatively few of the F_2 plants have been misclassified in these tests, thus it is expected that the heritability should be high. The heritability figures calculated for the progenies of Group 3, in which Manoa Wonder is involved, are lower, ranging from 73.4 to 77.6%. This is expected, since there is one less pair of genes segregating in these populations, so the genetic variance would be lower than in Group 1, but the environmental variance estimate would be about the same.

The lower heritability estimates observed in Groups 2 and 4 are due to the inclusion of some genetic variability from the apparently still segregating B 4096 in the environmental variance estimate. If the variance of the parent B 4096 is omitted as a component of the environmental variance in the heritability calculations, then the adjusted heritabilities become 74.3 and 62.3% in Group 2, and 53.3% in Group 4.

Table 19. Broad sense heritability estimates for Groups 1, 2, 3, and 4 calculated using parental F_1 and F_2 data

Parents	Broad sense heritability (%)
Group 1	
Harvester x PI 165426	89.2
Harvester x PI 226895	86.7
Harvester x 2114-12	91.4
Haw. Wonder x PI 165426	83.5
Haw. Wonder x PI 226895	85.1
Haw. Wonder x 2114-12	86.7
Group 2	
Harvester x B 4096	67.2
Haw. Wonder x B 4096	56.7
Group 3	
Man. Wonder x PI 165426	77.4
Man. Wonder x PI 226895	77.6
Man. Wonder x 2114-12	73.4
Group 4	
Man. Wonder x B 4096	52.3

Narrow sense heritability in F_3

The narrow sense heritability in the F_3 was estimated to be 68.3% on the basis of a parent-offspring regression of the mean scores of F_3 progenies on their F_2 parental plants (Figure 10, Regression line 1). This estimate, however, is probably lower than the true value. It is expected that 6 out of 7 plants in classes 1 and 5 are heterozygous and segregate in a 3:1 ratio, so that most class 1 plants produce progenies with class 2 plants and therefore means higher than 1.00, and most class 5 plants produce progenies with class 4 plants and means lower than 5.00. Similarly, class 2 and class 4 heterozygotes do not segregate in symmetrical ratios with the mean the same as the parent. Therefore, under this system of disease classification, the maximum heritability estimate would be significantly lower than 100%, even if the F_2 classification is completely correct. When only classes 2, 3, and 4 were used to calculate the regression coefficient, the heritability estimate increased to 79.1%, a considerable increase from 68.3% (Figure 10, Regression line 2). This high estimate of narrow sense heritability confirms the hypothesis of mostly additive gene action and the high accuracy of the evaluation method used.

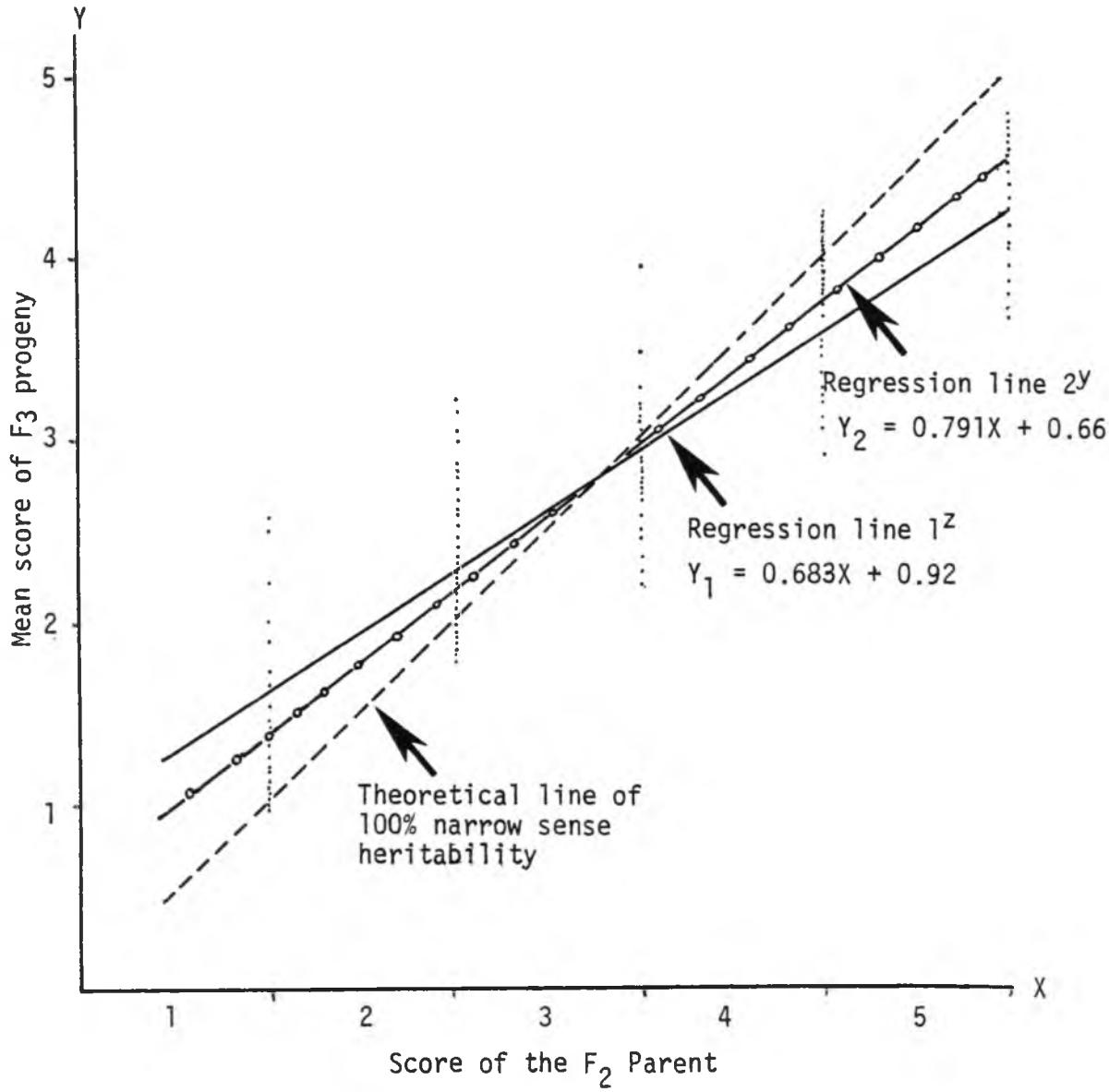


Figure 10. Regression of mean F_3 scores on the F_2 parental scores

[z]Line 1 - Regression Coefficient = 0.683 using all 5 classes

[y]Line 2 - Regression Coefficient = 0.791 using classes 2, 3, and 4

SUMMARY AND CONCLUSIONS

The crosses between the 2 types of resistant and 2 types of susceptible lines were treated in 4 separate groups. Group 1 includes the crosses between the 3 most resistant lines, Cornell 2114-12, PI 226895, and PI 165426, and the 2 most susceptible lines, Harvester and Hawaiian Wonder; Group 2 includes the crosses between the less resistant B 4096 and the 2 most susceptible lines; Group 3 includes the crosses between the 3 most resistant lines and the less susceptible Manoa Wonder; and Group 4 includes the crosses between the less resistant B 4096 and the less susceptible Manoa Wonder. Crosses among the resistant lines were considered a separate Group 5.

The F_1's of the crosses in Groups 1 to 4 were intermediate between the parents. The F_2 progeny segregated into 5 classes of susceptibility. There was no segregation in Group 5, indicating that the resistant parents probably carry the same genes for resistance. Reciprocal crosses responded similarly, so there was no evidence for cytoplasmic factors being involved. Crosses in Group 1 had homogeneous variances and therefore were combined, as were the crosses in Group 3. Groups 2 and 4, which had in common the resistant parent, B 4096, had heterogeneous variances and it was concluded that B 4096 is still segregating for resistance.

The segregations observed in Group 1 crosses fit a hypothesis that the 3 most resistant parents, Cornell 2114-12, PI 226895, and PI 165426, differ from the 2 most susceptible parents, Harvester and Hawaiian Wonder, by 3 gene pairs which are equal and additive in action. The

segregations observed in Group 3 crosses fit a hypothesis that the less susceptible Manoa Wonder is homozygous for one pair of genes for resistance and differs by only 2 pairs from Cornell 2114-12, PI 226895, and PI 165426. F_3 and F_4 progeny tests supported this hypothesis.

According to the 3 gene pair hypothesis, the following genotypes were assigned to different disease classes:

6 or 5 genes for resistance - Class 1 - Resistant

comprises two types
1. Homozygous
2. Heterozygous-segregate 3:1 into classes 1 and 2

4 genes for resistance - Class 2 - Still Resistant

comprises two types
1. Homozygous
2. Heterozygous-segregate 5:6:4:1 into classes 1, 2, 3, and 4

3 genes for resistance - Class 3 - Susceptible

comprises two types
1. Heterozygous-segregate 1:2:1 into classes 2, 3, and 4
2. Heterozygous-segregate 7:15:20:15:7 into all 5 classes

2 genes for resistance - Class 4 - Susceptible

comprises two types
1. Homozygous
2. Heterozygous-segregate 1:4:6:5 into classes 2, 3, 4, and 5

1 or 0 genes for resistance - Class 5 - Susceptible

comprises two types
1. Homozygous
2. Heterozygous-segregate 1:3 into classes 4 and 5

Broad sense heritability in the F_2 was estimated as between 73.4 and 91.4% under these controlled conditions. Narrow sense heritability in the F_3 was estimated to be more than 68.3%, which confirms that

genetic control was mostly by additive gene action. These results agree with those of Deaken and Dukes (1975) which showed that resistance is highly heritable and controlled by few genes, but disagree in not finding complete dominance as they reported. Here the resistance seems to be quantitatively inherited with additive gene action, more in agreement with the results of Dickson and Boettger (1977).

Testing for R. solani resistance in the field is probably nearly impossible because of variability of the pathogen population and also the presence of other pathogens which cause similar disease symptoms. The greenhouse screening procedure described in this study is a simple one which can be used to test a large number of seedlings at one time. The chances of escapes are minimized. Resistant individuals can be identified in 2 weeks, and selection for horticultural characteristics can be made in the same generation by transferring the resistant plants to the field.

APPENDIX

Table 20. Classification of F_1 progenies for *R. solani* infection

Parents	Infection Class and Frequency					Total	Mean	Difference
	1	2	3	4	5			
Group 1								
Harvester x PI 165426		4	13	1		18	2.83	0.00
PI 165426 x Harvester		1	5			6	2.83	
Harvester x PI 226895		3	10			13	2.76	0.37+
PI 226895 x Harvester		1	5	2		8	3.13	
Harvester x 2114-12			8	1		9	3.11	0.36+
2114-12 x Harvester		2	6			8	2.75	
Haw. Wonder x PI 165426		3	10	2		15	2.93	0.07+
PI 165426 x Haw. Wonder			4			4	3.00	
Haw. Wonder x PI 226895		3	7	1		11	2.82	0.18+
PI 226895 x Haw. Wonder		1	9	1		11	3.00	
Haw. Wonder x 2114-12		1	7	1		9	3.00	0.30+
2114-12 x Haw. Wonder		3	7			10	2.70	
Group 2								
Harvester x B 4096		2	7	6		15	3.27	0.02+
B 4096 x Harvester		1	4	3		8	3.25	
Haw. Wonder x B 4096		2	6	4		12	3.16	0.28+
B 4096 x Haw. Wonder		1	3	5		9	3.44	
Group 3								
Man. Wonder x PI 165426		9	3			12	2.25	0.25+
PI 165426 x Man. Wonder		4				4	2.00	
Man. Wonder x PI 226895		10	5			15	2.33	0.22+
PI 226895 x Man. Wonder		8	1			9	2.11	

+ Difference not significant, as determined by t test

Table 20. (continued) Classification of F_1 progenies for R. solani infection

Parents	Infection Class and Frequency 1	2	3	4	5	Total	Mean	Difference
Man. Wonder x 2114-12	2	9	1			12	1.92	0.08+
2114-12 x Man. Wonder	1	6	1			8	2.00	
Group 4								
Man. Wonder x B 4096		4	5	3		12	2.92	0.37+
B 4096 x Man. Wonder		1	3	3		7	3.29	
Group 5								
PI 165426 x B 4096	7	1				8	1.13	0.62+
B 4096 x PI 165426	2	1	1			4	1.75	
2114-12 x B 4096	5	5	2			12	1.75	0.42+
B 4096 x 2114-12	7	1	1			9	1.33	
PI 165426 x 2114-12	11	2				13	1.15	0.05+
2114-12 x PI 165426	8	2				10	1.20	
PI 226895 x 2114-12	8	3				11	1.27	-
PI 165426 x PI 226895	8	3				11	1.27	0.05+
PI 226895 x PI 165426	2	1				3	1.33	
PI 226895 x B 4096	1	2				3	1.66	0.17+
B 4096 x PI 226895	2	3	1			6	1.83	

+ Difference not significant, as determined by t test

Table 21. Classification of F_2 progenies for R. solani infection

Parents	Infection Class and Frequency					Total	Mean	Variance
	1	2	3	4	5			
Group 1								
Harvester x PI 165426	20	23	40	26	17	126	2.97	1.58
"	15	22	31	10	19	97	2.96	1.75
PI 165426 x Harvester	8	14	19	12	10	63	3.01	1.51
"	19	33	72	39	19	182	3.11	1.23
"	11	37	33	24	13	118	2.92	1.32
Harvester x PI 226895	16	42	61	15	18	152	2.85	1.26
"	9	13	28	24	15	89	3.26	1.29
PI 226895 x Harvester	9	21	29	18	6	83	2.92	1.18
"	6	15	21	9	8	58	3.00	1.32
Harvester x 2114-12	11	31	41	21	7	111	2.84	1.09
"	17	29	54	31	16	147	3.00	1.25
"	20	44	48	25	19	156	2.87	1.42
2114-12 x Harvester	5	11	21	9	8	58	3.00	0.89
"	12	20	36	22	16	106	3.09	1.48
Haw. Wonder x PI 165426	10	31	41	17	4	103	2.75	0.96
"	14	42	38	39	23	156	3.10	1.47
PI 165426 x Haw. Wonder	26	54	63	31	27	201	2.90	1.48
"	14	51	103	47	12	227	2.96	0.92

Table 21. (continued) Classification of F_2 progenies for R. solani infection

Parents	Infection Class and Frequency					Total	Mean	Variance
	1	2	3	4	5			
Haw. Wonder x PI 226895	13	26	24	15	14	92	2.90	1.62
"	7	11	18	16	6	58	3.05	1.38
PI 226895 x Haw. Wonder	14	21	21	15	8	79	2.77	1.56
"	10	11	26	18	4	69	2.92	1.24
Haw. Wonder x 2114-12	15	29	48	31	25	148	3.15	1.46
"	19	51	69	43	27	209	3.05	1.32
2114-12 x Haw. Wonder	8	9	28	15	11	71	3.07	1.39
Group 2								
Harvester x B 4096	6	18	30	16	11	81	3.10	1.26
"	1	4	28	17	9	59	3.49	0.81
"	6	12	34	21	15	88	3.30	1.25
B 4096 x Harvester	3	14	26	11	7	61	3.08	1.05
"	5	18	24	21	4	74	2.93	1.25
"	6	21	35	19	11	92	3.08	1.18
Haw. Wonder x B 4096	7	32	33	24	11	107	3.00	1.20
"	0	14	88	23	21	146	3.35	0.70
B 4096 x Haw. Wonder	1	11	37	13	9	71	3.25	0.84
"	8	23	49	15	11	106	2.98	1.07

Table 21. (continued) Classification of F_2 progenies for R. solani infection

Parents	Infection Class and Frequency					Total	Mean	Variance
	1	2	3	4	5			
Group 3								
Manoa Wonder x PI 165426	49	76	38	24	0	187	2.20	0.94
"	41	58	33	11	2	145	2.14	0.92
PI 165426 x Man. Wonder	31	74	32	21	6	165	2.37	1.05
"	39	69	31	17	3	159	2.22	0.99
Man. Wonder x PI 226895	41	57	27	20	2	147	2.22	1.08
"	59	79	41	24	9	212	2.27	1.23
PI 226895 x Man. Wonder	11	48	31	4	2	96	2.35	0.70
"	20	32	16	9	0	77	2.18	0.91
Man. Wonder x 2114-12	24	58	34	14	0	131	2.29	0.80
"	21	38	19	6	4	88	2.25	1.08
2114-12 " Man. Wonder	23	45	22	13	3	106	2.32	1.09
"	41	113	48	23	5	230	2.29	0.90
Group 4								
Man. Wonder x B 4096	19	44	67	32	9	171	2.81	1.07
"	32	89	43	15	0	179	2.23	0.71
"	41	96	53	30	4	224	2.38	0.96
B 4096 x Man. Wonder	16	48	81	36	14	195	2.91	1.15
"	21	63	31	6	13	134	2.45	1.23

Table 21. (continued) Classification of F_2 progenies for *R. solani* infection

Parents	Infection Class and Frequency 1	2	3	4	5	Total	Mean	Variance
Group 5								
PI 165426 x B 4096	50	10	3	-	-	63	1.21	0.27
B 4096 x PI 165426	89	21	2	-	-	112	1.22	0.22
B 4096 x 2114-12	46	39	3	-	-	88	1.51	0.32
2114-12 x B 4096	43	6	3	-	-	52	1.23	0.27
PI 165426 x 2114-12	156	21	-	-	-	177	1.12	0.12
2114-12 x PI 165426	102	17	1	-	-	120	1.16	0.15
"	38	3	-	-	-	41	1.07	0.04
PI 226895 x 2114-12	34	7	1	-	-	42	1.21	0.22
"	42	6	-	-	-	48	1.13	0.11
PI 165426 x PI 226895	91	6	-	-	-	97	1.06	0.15
PI 226895 x B 4096	39	11	6	-	-	56	1.41	0.46

Table 22. Testing F_2 segregation ratios of group 1(most resistant vs. most susceptible) crosses.

Cross	Infection Class					Total	Chi Square			
	1	2	3	4	5		1:4:6:4:1 ratio	7:15:20:15:7 ratio	5:11 ratio	22:42 ratio
Harvester x PI 165426	20	23	40	26	17	126	29.16**	4.79	0.59	0.12
"	15	22	31	10	19	97	50.83**	14.69**	2.36	0.73
PI 165426 x Harvester	8	14	19	12	10	63	15.29**	2.14	0.28	0.10
"	19	33	72	39	19	182	16.61**	6.79	0.64	3.26
"	11	37	33	24	13	118	14.98**	4.20	4.76*	1.83
Harvester x PI 226895	16	42	61	15	18	152	24.62**	16.88**	3.04	1.05
"	9	13	28	24	15	89	19.62**	26.07**	1.87	3.82
PI 226895 x Harvester	9	21	29	18	6	83	4.11	1.61	0.87	0.10
"	6	15	21	9	8	58	7.44	2.70	0.73	0.08
Harvester x 2114-12	11	31	41	21	7	111	5.06	5.11	2.04	0.64
"	17	29	54	31	16	147	17.11**	2.45	0.10	0.75
"	20	44	48	25	19	156	26.71**	5.99	6.69**	2.78
2114-12 x Harvester	5	11	21	9	8	58	13.85**	3.31	0.10	0.73
"	12	20	36	22	16	106	18.25**	2.97	0.07	1.03

Table 22. (Continued) Testing F_2 segregation ratios of group 1(most resist. vs. most suscept.) crosses.

Cross	Infection Class					Total	Chi Square			
	1	2	3	4	5		1:4:6:4:1 ratio	7:15:20:15:7 ratio	5:11 ratio	22:42 ratio
Haw. Wonder x PI 165426	10	31	41	17	4	103	7.51	11.16*	3.67	1.56
"	14	42	38	39	23	156	17.69**	5.90	1.46	0.11
PI 165426 x Haw. Wonder	26	54	63	31	27	201	37153**	8.35	6.67**	2.67
"	14	51	103	47	12	227	6.71	26.77**	0.73	3.30
Haw. Wonder x PI 226895	13	26	24	15	14	92	25.46**	6.02	5.03*	2.34
"	7	11	18	16	6	58	5.11	1.09	0.60	0.30
PI 226895 x Haw. Wonder	14	21	21	15	8	79	22.00**	4.32	5.85*	3.60
"	10	11	26	18	4	69	15.60**	5.04	0.67	0.57
Haw. Wonder x 2114-12	15	29	48	31	25	148	36.03**	5.63	1.12	1.46
"	19	51	69	43	27	209	20.51**	3.02	0.55	0.10
2114-12 x Haw. Wonder	8	9	28	15	11	71	17.03**	6.76	1.84	3.08

*Significant at 5% level
**Significant at 1% level

Table 23. Testing F_2 segregation ratio of Group 3 (most resistant x less susceptible) individual progenies

Parents	Infection Class and Frequency					Total	Chi-square 11:5 Ratio
	1	2	3	4	5		
Man. Wonder x PI 165426	49	76	38	24	0	187	0.39
"	41	58	33	11	2	145	0.13
PI 165426 x PI 226895	31	74	32	21	6	165	1.69
"	39	69	31	17	3	159	0.30
Man. Wonder x PI 226895	41	57	27	20	2	147	0.28
"	59	79	41	24	9	212	1.81
PI 226895 x Man. Wonder	11	48	31	4	2	96	2.37
"	20	32	16	9	0	77	0.36
Man. Wonder x 2114-12	24	58	34	14	0	131	1.74
"	21	38	19	6	4	88	0.20
2114-12 x Man. Wonder	23	45	22	13	3	106	1.41
"	41	113	48	23	5	230	1.32

Table 24. Testing F_2 segregation ratio of Groups 2 and 4 crosses with B 4096 as resistant parent

Parents	Infection Class and Frequency					Chi-square		
	1	2	3	4	5	5:11 ratio	22:42 ratio	11:5 ratio
Group 2								
Harvester x B 4096	6	18	30	16	11	0.11	0.87	-
"	1	4	28	17	9	16.01**	13.51**	-
"	6	12	34	21	15	5.23*	7.28**	-
B 4096 x Harvester	3	14	26	11	7	0.31	1.16	-
"	5	18	24	21	4	1.15	0.24	-
"	6	21	35	19	11	0.20	1.19	-
Haw. Wonder x B 4096	7	32	33	24	11	0.70	0.10	-
"	0	14	88	23	21	32.50**	39.42**	-
B 4096 x Haw. Wonder	1	11	37	13	9	6.58**	9.06**	-
"	8	23	49	15	11	0.18	1.05	-
Group 4								
Man. Wonder x B 4096	19	44	67	32	9	2.73	0.41	82.71**
"	32	89	43	15	0	109.79**	85.87**	0.10
"	41	96	53	30	4	93.27**	71.24**	6.01*
B 4096 x Man. Wonder	16	48	81	36	14	0.21	0.20	120.52**
"	21	63	31	6	13	47.98**	61.38**	2.21

* Significant at 5% level
**Significant at 1% level

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